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Archives of Neurology and Psychiatry

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SENSORY CHANGES IN THE SUBACUTE COMBINED DEGENERATION OF PERNICIOUS ANEMIA *

ARTHUR S. HAMILTON, M.D. AND CHARLES E. NIXON, M.D.
MINNEAPOLIS

In the following article we desire to summarize the development of our knowledge of sensory phenomena associated with pernicious anemia and to record the sensory changes encountered in forty-one cases of pernicious anemia studied in the Elliott Memorial Hospital of the University of Minnesota and in the university service of the Minneapolis General Hospital. Nearly all these cases were also studied by the members of Dr. Rowntree's staff in internal medicine and no case appears in our list in which the diagnosis of pernicious anemia was disputed. Most of our cases belong to the Addison-Biermer type of pernicious anemia.

HISTORICAL SURVEY

Addison [1] (1855), in his original description of pernicious anemia, said nothing of involvement of the nervous system except that he found some fatty degeneration of a portion of the semilunar ganglion of the solar plexus.

Biermer [2] (1872) referred to: weakness, giddiness and palpitation and also reported capillary hemorrhages in the brain and the retina.

In 1880, Herbert W. Little [3] stated: "The indications of the nervous affection in this disease are: frequent cephalalgia, vertigo, syncope, neuralgic pains, dyspepsia, vomiting, diarrhea, mental depression, muscular weakness, pallor of the skin, reduction or rise of temperature." The author thought the nervous system the primary seat of trouble and involvement of the vasomotor system important.

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* From the Division of Nervous and Mental Diseases and the Neuropathological Laboratory, University of Minnesota Medical School.

1. Addison, T.: On the Constitutional and Local Effects of Disease of the Suprarenal Capsule, 1855.

2. Biermer: Progressive perniciöse Anaemie, Cor.-Bl. f. schweiz. Aerzte **2**: No. 1, 1872.

3. Little, H. W.: Progressive Pernicious Anemia, a Disease of the Vasomotor System, Med. Rec. **17**:313, 1880.

E.K.N. 1-24-22

In 1884, Lichtenstern [4] reported two cases of pernicious anemia in tabetics. It is probable that these were not cases of tabes but of pernicious anemia. Lichtenstern referred to posterior column degeneration, but said nothing of sensory changes.

In 1887, Lichtheim [5] reported three cases of anemia; one patient had stiffness of the legs, a feeling of weakness, paresthesia, ataxia and the Romberg sign. There was widespread degeneration of the columns of Goll and Burdach, but of the latter a small rim remained intact. Foci of degeneration were found in the lateral and anterior columns. The peripheral nerves were not affected.

In Putnam's [6] (1891) eight cases of system sclerosis, the nerve roots were more or less altered in the four in which pathologic studies were made, the anterior somewhat more than the posterior. In all of his cases, numbness was a pronounced feature, lancinating pains in only one. One patient complained of a sense of constriction around the chest and abdomen and that at times she felt "as if her stockings were filled with pebbles." Objective impairment of muscle sense and diminished cutaneous sensibility were observed in all but one case.

Dana [7] (1891) reported a patient with a sensation of pins and needles in her heels and queer, indescribable, somewhat painful feelings in both lower extremities with every change of weather. There was no cutaneous anesthesia. The posterior roots in the lumbar region showed decided degenerative changes.

Grainger Stewart [8] (1891) reported a case which he believed conformed to the Putnam-Dana type of combined sclerosis. There was painful tingling in the upper and lower extremities. A year later the paresthesias were more marked and sensibility was diminished.

Von Noorden [9] (1891-1892) found paresthesias in the extremities and impaired sensibility in the feet, and noted an intact area next the posterior roots though the posterior columns were greatly degenerated.

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4. Lichtenstern, O.: Progressive perniciöse Anämie bei Tabeskranken, Deutsch. med. Wchnschr. **10**:849, 1884.

5. Lichtheim: Zur Kenntniss der perniciösen Anämie, Verhandl. d. Cong. f. inn. Med. **6**:84-99, 1887.

6. Putnam: A Group of Cases of System Sclerosis of the Spinal Cord, Associated with Diffuse Collateral Degeneration Occurring in Enfeebled Persons Past Middle Life, and Especially in Women; Studied with Particular Reference to Etiology, J. Nerv. & Ment. Dis. **16**: (Feb.) 1891.

7. Dana, C. L.: Subacute Combined Sclerosis of the Spinal Cord and Its Relation to Anemia and Toxemia, J. Nerv. Ment. Dis. **26**:1, 1889; 1891, February and April.

8. Stewart, Grainger: Notes on a Case Probably Referable to a Recently Described Form of Spinal Disease, Brit. M. J. **1**:1219, 1891.

9. Von Noorden: Untersuchung über schwere anämie, Charité Ann., 1891, p. 217.

Eisenlohr [10] (1892) described a case with itching; there was degeneration of the right saphenous nerve and subacute combined degeneration of the cord.

Minnich [11] (1892) reported six cases of pernicious anemia, in which all the patients had objective sensory disturbances, increasing toward the periphery, such as loss of position sense in toes, lowered pain sensibility, especially in the peripheral parts, but not so involved as tactile sense, together with impaired position sense and some impairment of the sense of pressure over the tibia. The cords in these cases showed the usual patchy degeneration in Burdach's column and sharply defined areas of degeneration in Goll's column. The posterior roots and the peripheral nerves, where examined, were normal.

Nonne [12] (1893) reported two cases of pernicious anemia with numbness of the hands and feet and girdle sensation. In one case the numbness was present long before the anemia was obvious. There were some hypalgesia and hypesthesia. In a report [13] (1895) of seventeen cases of pernicious anemia with cord changes he noted sensory disturbances in four. The peripheral nerves were examined in three and no noteworthy degeneration found. With the Weigert method, the posterior roots and root entrance zones appeared practically intact, but with the Marchi method there was some degeneration of the posterior roots.

Burr [14] (1894) stated that though paresthesias are frequent and delayed or diminished sensation not infrequent, absolute anesthesia almost never occurs. He comments on the marked discrepancy between the severe anemia and the normal cord in one case. The peripheral nerves in one case were normal.

Boedeker and Juliusberger [15] (1896) reported the case of a patient with pain in the entire body, especially the bones, and a sense of dead-

10. Eisenlohr: Ueber primäre Atrophie der Magen- und Darmschleimhaut und deren Beziehung zu schwerer Anämie und Rückenmarkserkrankung, Deutsch. med. Wchnschr. **49**:1105, 1892.

11. Minnich, W.: Zur Kenntniss der im Verlauf der perniciösen Anämie beobachteten spinal Erkrankungen, Ztschr. f. klin. Med. **21**:25-60, 264-314, 1893; **22**:60-86, 1893.

12. Nonne, M.: Beiträge zur Kenntniss der im Verlaufe der perniciöser Anämie beobachteten Spinalerkrankungen, Arch. f. Psychiat. **25**:421-449, 1893.

13. Nonne, M.: Weitere Beiträge zur Kenntniss der im Verlaufe letaler Anämien beobachteten Spinalerkrankungen, Deutsch. Ztschr. f. Nervenheilk. **6**:313-368, 1894-1895.

14. Burr, C. W.: The Spinal Cord Lesion and Symptoms of Pernicious Anemia, Univ. Med. Mag. **7**:472-491, 1894.

15. Boedeker and Juliusberger: Demonstration von Rückensmarksveränderungen bei tötlicher Anämie, Centralbl. f. Nervenheilk u. Psychiat. **7**:315-318, 1896.

ness in the feet. In a second patient there was a feeling of deadness in the legs, coldness and numbness in the fingers and later a hyperesthesia of the entire body and tearing pains in the legs. Three months later there was disturbance of the muscle sense with practically intact skin sensibility and two months after that, a feeling of deadness in the legs with essentially still intact skin sensibility, including temperature sense. One month later the muscle sense was practically gone. Skin sensibility was lost up to the neck. There was sclerosis of the middle root zone but especially of Goll's column and also of a part of the root entrance zone. The extramedullary roots and peripheral nerves were intact.

Lloyd's [16] (1896) patient complained of cold sensations running up and down his legs, of pricklings in the same regions and of a feeling of walking on cotton; also girdle sensations about the abdomen. The lesion in the posterior columns was about midway between the columns of Goll and Burdach, extending not quite to the posterior commissure in front, nor quite to the periphery of the cord behind. The posterior root zone was not seriously involved. The nerve trunks showed some change with the Weigert stain.

Bastianelli [17] (1896) reported a group of patients with subacute combined degeneration symptoms developing first or coincidental with the anemia, which is of secondary importance. These cases he seems to set aside from true subacute combined degeneration of pernicious anemia, but it is probable that in at least some the symptoms of pernicious anemia would appear later.

Petren [18] (1896) found loss of touch, temperature and muscle sense in all the extremities, especially the legs, in one of his patients.

Dana [19] (1899) referred to the prominent and persistent subjective sensory phenomena and found diminution of touch, pain and temperature sense.

Russell, Batten and Collier [20] (1900) found girdle sensation and severe lightning pains to be frequent. Anesthesia of the lower extremi-

16. Lloyd, J. H.: The Spinal Cord in Pernicious Anemia, J. Nerv. & Ment. Dis. **21**:225-235, 1896.

17. Bastianelli, G.: Le sclerosi combinati del midollo spinale nelle anemie perniciosi, Bull. de roy. Acad. de med. di Roma **22**:197-244, 1896-1897; Ref. in Neurol. Centralbl. **16**:78, 1897.

18. Petren, K.: Bidrag till Kännedomen om Ryggmärgsförändringar vid Perniciös Anämie, Nord. Med. Arkiv. **6**:71, 1896; Ref. Neurol. Centralbl. **15**:747, 1896.

19. Dana, C. L.: Subacute Ataxic Paraplegia and Combined Sclerosis—a Form of Spinal Disease Associated with Lethal Anemia and Toxemia, Med. Rec. **55**:897-902, 1899.

20. Russell, J. S. R.; Batten, F. E., and Collier, J.. Subacute Combined Degeneration of the Spinal Cord, Brain **22**:39-110, 1900.

ties and lower trunk rapidly becomes absolute and rises segment after segment.

Billings [21] (1902) reported thirty-six cases of subacute combined degeneration with pernicious anemia, in all of which paresthesia occurred early. In twenty-four the sensory changes were subjective only: tingling, numbness, coldness, formication, heat, etc. Hyperesthesia occurred in one case. Girdle sensation was present in all developing spasticity. The subjective symptoms were usually stationary, but disappeared in one case. In some there was complete loss of

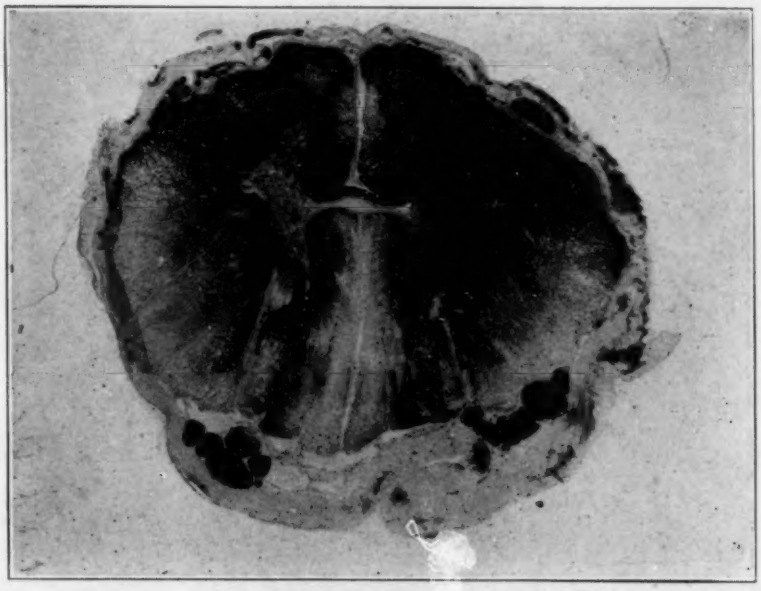

Fig. 1 (Case 2).—Third cervical segment cord; Weigert stain.

sense of position even though touch and temperature were only slightly involved. The column of Lissauer and the posterior roots were intact.

J. M. Clark [22] (1904) stated that the most prominent clinical symptoms are paresthesias of various kinds. A common complaint was of numbness or deadness of the fingers which rendered fine movements difficult. These paresthesias vary much in intensity during the course of the illness. Objective disturbance of sensibility is not marked, but

21. Billings, F.: The Changes in the Spinal Cord and Medulla in Pernicious Anemia, Boston M. & S. J. **147**:225-233, 257-263, 1902.

22. Clark, J. M.: On the Spinal Cord Degenerations in Anemia, Brain **28**:441-459, 1904.

in the later stages may consist of some deficient appreciation for all forms of sensation over the lower extremities. In one case examined the peripheral nerves were healthy.

Putnam and Taylor [23] (1901) reported some form of paresthesia as strikingly prominent and usually felt first in the feet and then in the hands, either in all the fingers alike or in the median or ulnar areas by preference. The proximal segments of the shoulder or thigh were at times attacked early, if not first, as occasionally in tabes.

Bramwell [24] (1915) insisted particularly on the occurrence of cord symptoms which may long antedate positive signs of pernicious

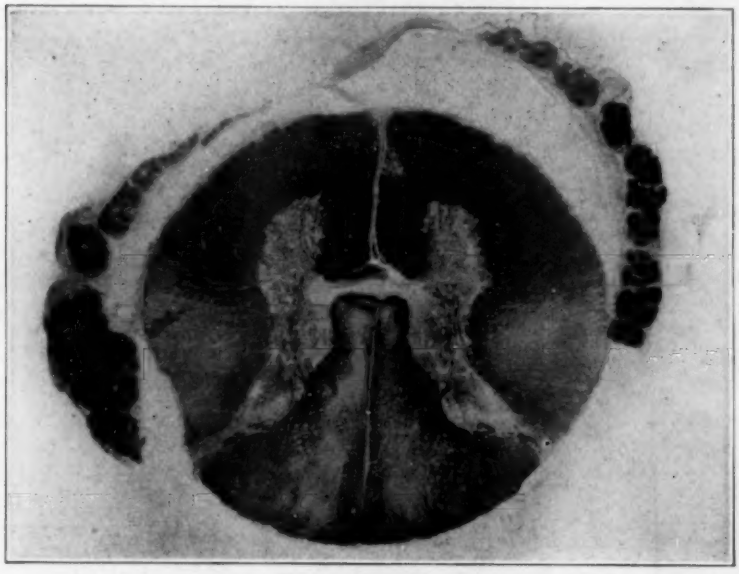

Fig. 2 (Case 2).—Twelfth thoracic segment cord; Weigert stain.

anemia—in one about three years. Then rapid destruction of blood takes place. In one patient there were shooting pains in the legs, numbness and tingling in the feet and a cold feeling in the knees and elbows. Bramwell also reemphasized the stocking and glove distribution of the sensory loss.

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23. Putnam, J. J., and Taylor, E. W.: Diffused Degeneration of the Spinal Cord, J. Nerv. & Ment. Dis., **28**:1-21, 1901.

24. Bramwell, B.: On the Association of Pernicious Anemia with Subacute Combined Degeneration of the Spinal Cord, Edinburgh M. J. **14**:260-276, 1915.

In 1913 and 1914, J. Déjerine, A. Déjerine, Crouzon and Jumentie,[25] were the first clearly to point out the early and marked development of loss of deep sensibility in subacute combined degeneration. In the first publication, Déjerine divided sclerosis of the posterior and lateral columns into two groups: (1) classic tabes complicated with involvement of the lateral columns. In this type the sensory changes are a marked alteration of the superficial senses. There is a slowing rather than a diminution in the intensity of the perception of the latter. There exists also extensive alteration of deep sensibility. (2) A second variety of combined sclerosis of subacute course shows no signs of syphilis, and the condition is rather associated with pernicious anemia.

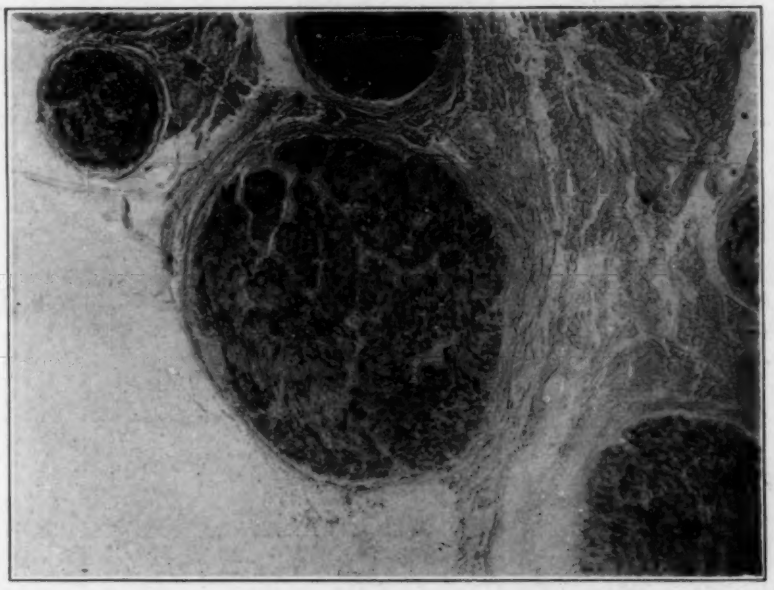

Fig. 3 (Case 2).—Peripheral nerve; Weigert stain.

There is integrity of all kinds of superficial sensibility, tactile in particular, while deep sensibility as shown in sense of position, bone, deep pressure and stereognosis, is abolished.

The explanation of this variation lies in the difference in the topography. In the tabetic process, the sclerosis is of radicular origin. The external part of the column of Burdach, the posterior horn and the

25. Déjerine, J.: Le syndrome des fibres radiculaires longues des cordons postérieurs, Compt, rend. Soc. de biol. **75**:554-556, 1913. Déjerine, A. and Mouzon, J.: Contribution a l'etude du syndrome des fibres radiculaires longues des cordons postérieurs dans l'anémies pernicieuse, Rev. neurol. **22**:382-388, 1914. Déjerine, J. and Jumentie, J.: Un cas de syndrome des fibres radiculaires longues des cordons postérieurs, suivi d'autopsie, Rev. neurol., ibid., 271-273.

posterior roots are always involved, and since the external part of Burdach is the point of passage through the cord of the short fibers of the posterior roots, conductors of tactile, pain and temperature sense, these are naturally involved. In subacute combined sclerosis, the posterior roots and the external part of the column of Burdach are always intact, and only the long fibers of the columns of Goll and the adjacent part of the column of Burdach are involved. Since the different kinds of deep sensibility pass only by the long fibers of the posterior columns, these sensibilities are earliest and most involved in subacute combined degeneration of the cord. Déjerine has called integrity of

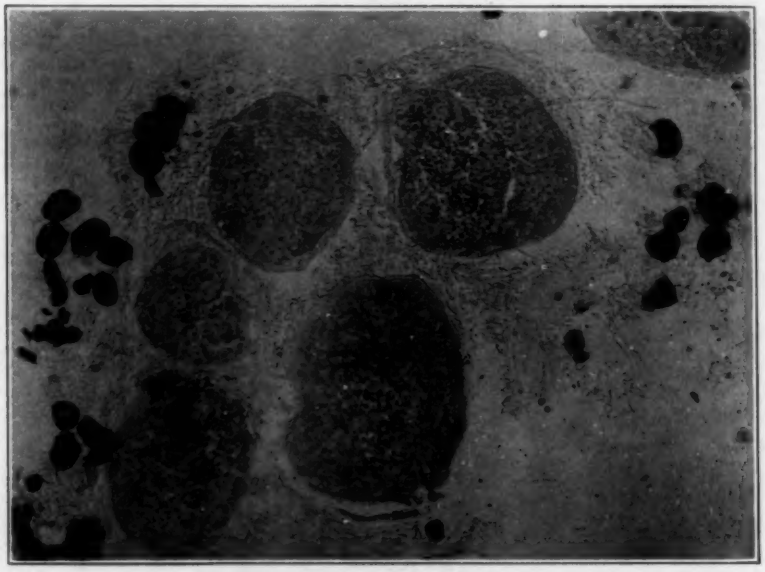

Fig. 4 (Case 2).—Peripheral nerve; Marchi stain.

superficial sensibility with marked disturbance of deep sensibility the syndrome of the long radicular fibers of the posterior columns.

Cadwalader [26] (1916) reported eight cases recently observed by himself. All complained of subjective disturbances of sensation. Tactile, pain and temperature sensibility were normal in all but one case, while vibration sensibility and sense of position were diminished or lacking in all.

Schaller [27] (1918) concluded that "the cord changes in combined sclerosis are pseudo-system degenerations of toxic type." He explains

26. Cadwalader, W. B.: Diagnosis of Subacute Combined Sclerosis of the Spinal Cord Associated with Pernicious Anemia, J. A. M. A. **66**:2035-2036, 1916.

27. Schaller, W. F.: Spinal Cord Changes in Combined Sclerosis, California State J. M. **16**:44, 1918.

the early involvement of deep sensation and the conservation of superficial sensation by the localization of the lesions.

Woltmann [28] (1919) reported 150 cases of pernicious anemia from the Mayo Clinic. Vibration or joint sensibility was impaired in 92 per cent. of the patients showing involvement of the nervous system, whereas superficial sensibility was impaired in only 42.4 per cent. of their cases.

SENSORY PHENOMENA

Frequency of Sensory Phenomena in Pernicious Anemia.—Authors vary widely in their opinions. McCrae [29] reported forty cases of pernicious anemia; ten patients showed nervous manifestations by examination, but only four complained of definite nervous troubles. McPhedran [30] found symptoms of disease of the nervous system in nine out of twenty-two patients and Woltmann in 80.6 per cent. of his 150 patients.

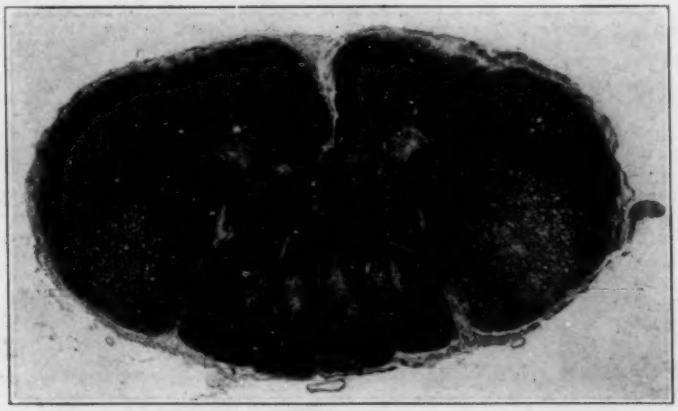

Fig. 5 (Case 3).—Seventh thoracic segment cord; Weigert stain.

Subjective Sensory Phenomena.—Subjective sensory disturbances are so common and so pronounced in subacute combined degeneration, as seen by the foregoing, that they have come to be looked on as most important diagnostic phenomena, and by some are considered so characteristic as almost in themselves to constitute a sufficient basis for diagnosis. The most frequent sensory disturbances are: tingling, numbness, sensation of deadness, stiffness, tightness, heat or cold or of something trickling or creeping and shooting pains.

28. Woltmann, H. W.: Nervous Symptoms in Pernicious Anemia, Am. J. M. Sc. **157**:400 (March) 1919.

29. McCrae, T.: Pernicious Anemia. The Statistics of a Series of Forty Cases, J. A. M. A. **28**:148-150, 1902.

30. McPhedran, A.: Observations on the Nature and Treatment of Pernicious Anemia, Lancet **1**:148-150, 1902.

These sensations are at first on the periphery of the limbs, usually in a stocking and glove fashion, more rarely in a nerve root distribution. They have been reported as appearing in the tongue. Subsequently they spread toward the trunk, and are followed at a variable interval by sensory loss.

Objective Sensory Phenomena.—From the time of Putnam [6] (1891) objective sensory phenomena have been noted, but there is little specific information as to the type of sensory loss, until the report of Minnich,[11] who states that there is a lowering of all forms of sensibility, increasing toward the periphery, with tactile sense more involved than pain and with sense of position lost in the toes. Later observers reported various

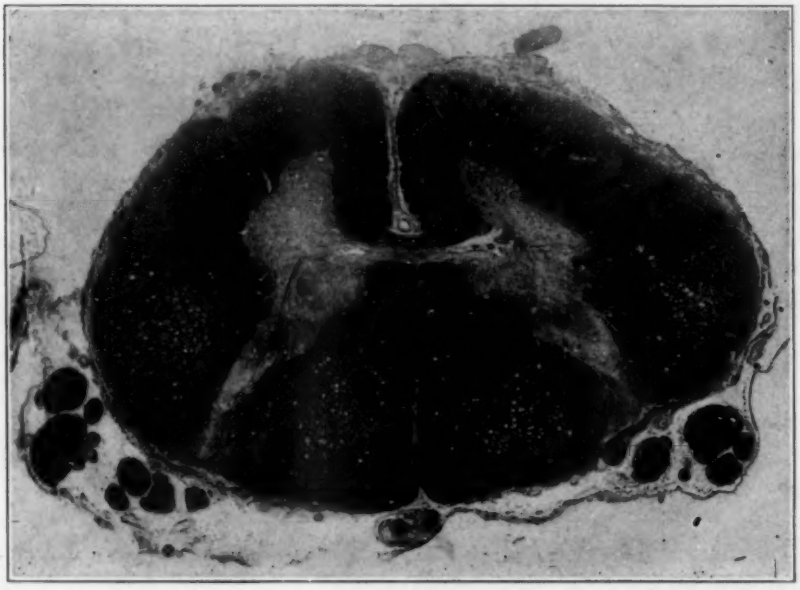

Fig. 6 (Case 3).—First lumbar segment cord; Weigert stain.

degrees of anesthesia and analgesia, loss of sense of position and impaired localization.

Russell, Batten and Collier [20] spoke of a considerable loss of sensibility of all forms in the legs and lower part of the trunk with sense of passive position lost in the lower extremities and much impaired in the hands. Other forms of sensibility were impaired equally and in an increasing degree. Anesthesia of the skin was always peripheral first, appearing over the feet and hands, but as it gradually extended upward it became segmental in distribution, and its upper limit on the trunk was always a segmental limit, sharply defined. The highest level they observed was the upper limit of the sixth cervical root area. In its

progress upward it may be limited for a long time to a certain segmental level and then suddenly encroach for several segments higher and over the newly involved area may show marked fluctuations in its progress to complete anesthesia. Anesthesia dolorosa and delay amounting to as much as fifteen seconds have been encountered. The lower lumbar and the first sacral areas were always affected before the lower sacral and upper lumbar areas, and the lower cervical segments before the upper thoracic segments. They state that microscopically even in extreme cases there is a narrow band of normal tissue just inside the posterior horns and that the column of Goll is more affected than that of Burdach.

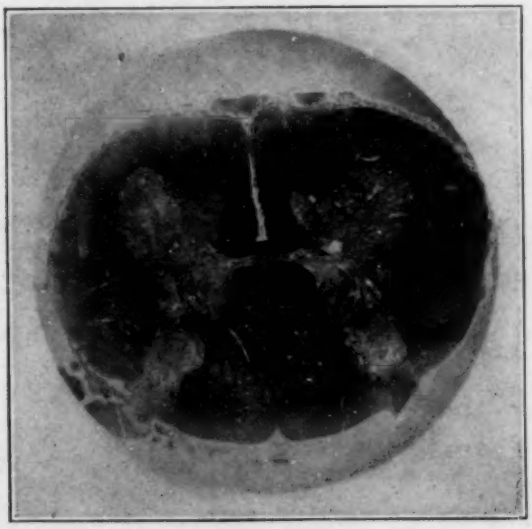

Fig. 7 (Case 3).—Second lumbar segment cord; Weigert stain.

Gordon [31] reported a case with a sensory syringomyelic dissociation in the upper extremities.

Foster Kennedy [32] reported a case with some diminution of pain and tactile sensation of irregular distribution on the legs. Temperature sense and deep muscle pain sense were normal, but there was marked loss of sense of position in feet and hands.

Hyperthesia has been noted occasionally, among others by Boedeker and Juliusberger,[15] Billings,[21] Nonne,[12] Minnich,[11] and

31. Gordon, A.: Histological Changes of the Spinal Cord in Pernicious Anemia Apropos of a Case with Diffused Degeneration, New York M. J. **90**:8-12, 1909.

32. Kennedy, Foster: Subacute Combined Degeneration of the Spinal Cord Associated with Anemia, Med. Rec. **84**:84, 1913.

Thompson.[33] In one of our cases there was marked hyperesthesia throughout a period of two years; the patient objected strenuously to a blanket on her feet and legs. During an attack of anasarca, this hyperesthesia disappeared and did not return.

RELATION OF SEVERITY OF BLOOD PHENOMENA TO NERVOUS PHENOMENA

Lack of parallelism in the severity of the blood and nervous phenomena has been noted by many writers. On the basis of this lack of congruence, Bastianelli [17] divided his cases into two groups. In the first group anemia is the predominating symptom, the cord symptoms

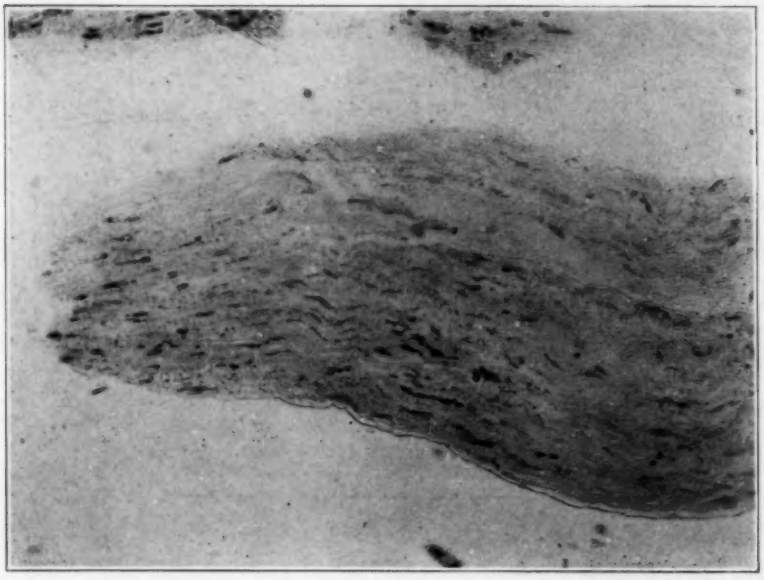

Fig. 8 (Case 3).—Posterior roots; Marchi stain.

developing later and in mild degree. In the second group nervous symptoms develop first or coincidently with the anemia, and the nervous symptoms predominate throughout.

J. M. Clark [22] said the anemia may (*a*) precede the cord changes; (*b*) it may accompany them from the first, and proceed pari passu with them; or (*c*) it may appear late in the course of the cord affection, and his statement is supported by the cases of a few other writers.

33. Thompson, T.: A Case of Combined Degeneration of the Spinal Cord Demonstrating the Nature of Afferent Impulses in the Posterior Columns, Brain **34**:510, 1911-12.

Siemerling[34] reported a case in which there was improvement in the blood picture with simultaneous progression of the cord involvement.

While receiving high caloric feeding the hemoglobin of one of our patients (W. G.) rose from 40 to 80 per cent. and the red cell count from 1,080,000 to over 3,000,000. This man previously had shown no impairment of vibration sense, yet during this improvement in the blood, accompanied by improvement in all his symptoms except the subjective sensory disturbances, he showed diminished appreciation of vibration for the first time.

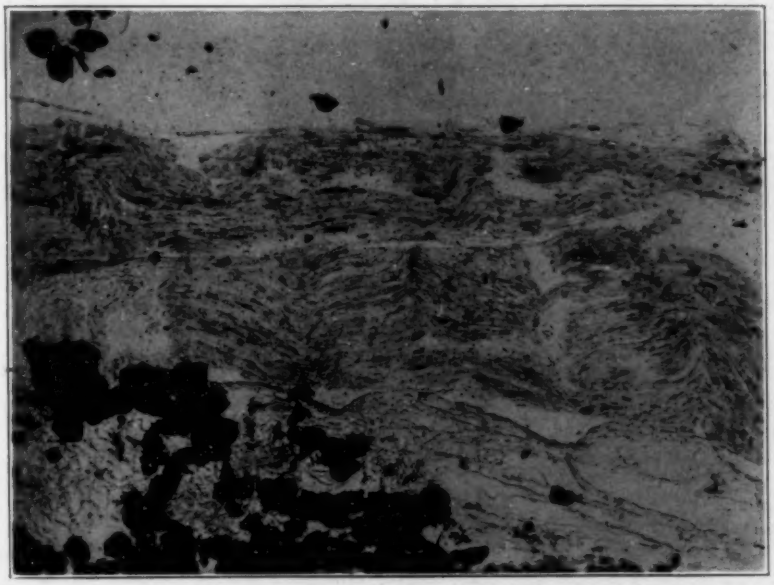

Fig. 9 (Case 4).—Peripheral nerve; Marchi stain.

INCONGRUENCE OF PATHOLOGIC AND CLINICAL FINDINGS

A similar lack of parallelism at times appears between the extent of the pathologic changes and the severity of the symptoms. Minnich and Nonne offer, as a partial explanation of this disharmony, the special location of the lesions in the posterior columns.

One of our patients (U. H. No. 12082), with a severe anemia, paresthesia and absence of vibration sense in the toes and marked impairment in the malleoli, showed no definite findings in the cord; the peripheral nerves, however, were distinctly involved. In another patient (U. H. No. 13141) with severe anemia, some numbness and

34. Siemerling, E.: Rückenmarkserkrankungen und Psychose bei perniciöser Anämie, Arch. f. Psychiat. **45**:567-594, 1909.

tingling in the feet and with vibration sense lost in the toes and much impaired in the malleoli, there was no change in the cord except a possible pallor along the dorsal septum, but there was marked degeneration of the peripheral nerves.

OPTIC NEURITIS

Optic neuritis is referred to by many writers, but either is not frequently sought or is not often present. It occurred only once in fifty-eight of Collier's cases, and even in this instance was probably of syphilitic origin. However, von Noorden and others have reported eyeground change of some sort as occurring frequently in severe anemias. Additional cases of optic neuritis are reported by Boedeker and Juliusberger,[15] Russell,[35] Hawthorne,[36] Bramwell[37] and Wolt-

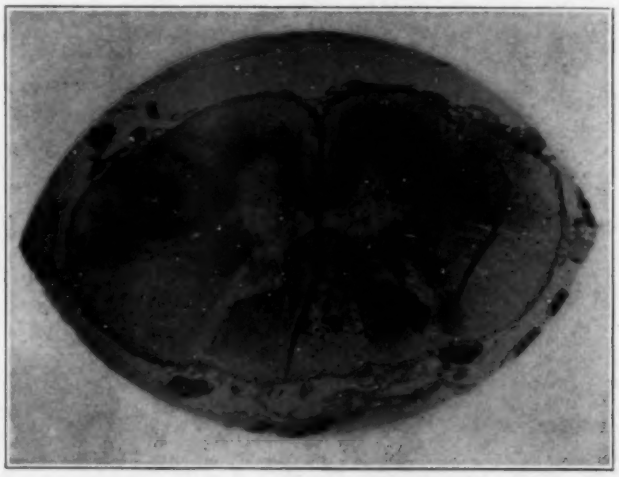

Fig. 10 (Case 5).—Eleventh thoracic segment cord; Weigert stain.

mann.[28] Bielschowsky found changes in the optic nerve in two cases. Schroeder found hemorrhage near the macula lutea in one case and Bastianelli[17] found optic nerve atrophy, confirmed at necropsy, in a case of pernicious anemia with spinal sclerosis; in the same case the posterior roots were intact. Putnam and Taylor[23] found optic atrophy in two and probably three out of fifty cases.

35. Russell, R.: The Relationship of Some Forms of Combined Degeneration of the Spinal Cord to One Another and to Anemia, Lancet **2**:4, 1898.

36. Hawthorne, C. O.: An Address on the Cerebral and Ocular Complications of Anemia and the Probable Relationship of these to Thrombosis, Lancet **2**:857-863, 1908.

37. Bramwell, Byron: Remarks on a Case of Subacute Combined Degeneration of the Spinal Cord Simulating Disseminated Sclerosis, Brit. M. J. **6**:1396, 1910.

THE PERIPHERAL NERVES

In most of the cases reported it is evident that the peripheral nerves have been studied either insufficiently or not at all. When studied the results have varied greatly.

Lichtheim [5] reported the peripheral nerves intact.

Von Noorden,[38] in 1892, stated that the alterations resembled those of a multiple neuritis with carcinoma. In his case, the crural nerve showed no definite changes but both tibials were markedly degenerated, two-fifths to three-fourths of the nerve fibers showing changes.

Nonne [13] particularly notes that the posterior roots were practically intact. In three of his cases the peripheral nerves were studied and showed no noteworthy degeneration.

Fig. 11 (Case 7).—Eleventh thoracic segment cord; Weigert stain.

Burr [14] reported in one case slight degeneration in the posterior roots, but Lissauer's zone and the peripheral nerves were intact. Bowman,[39] Billings,[21] Hughes and Spiller [40] found Lissauer's zone and the posterior roots intact, Rothmann [41] found slight degeneration in

38. Von Noorden, Carl: Untersuchung über schwere Anämie, Charité Annalen, 1892, p. 217.

39. Bowman, H. M.: On the Association of Diseases of the Spinal Cord with Pernicious Anemia, Brain **17**:198-213, 1894.

40. Hughes, W. E. and Spiller, W. G.: Report of a Case of Pernicious Anemia with Changes in the Spinal Cord, J. Nerv. & Ment. Dis. **28**:573-574, 1901.

41. Rothmann, M.: Die primären combinierten Strangerkrankungen des Rückenmarks, Deutsch. Ztschr. f. Nervenkrankh. **7**:171-262, 1895.

places in the posterior roots. Boedeker and Juliusberger [15] found the extra medullary roots and the peripheral nerves intact though there was some degeneration of the root entrance zone. Lloyd [16] found some changes in the nerve trunks, the fibers not staining evenly with Weigert's method.

Russell, Batten and Collier [20] found that usually Lissauer's tract appears normal. Various peripheral nerves were examined and, though usually normal, occasionally slight changes were found with the Marchi or Weigert-Pal method. In one case, however, there was considerable degeneration in both the larger and smaller nerve fibers, interpreted as of late origin.

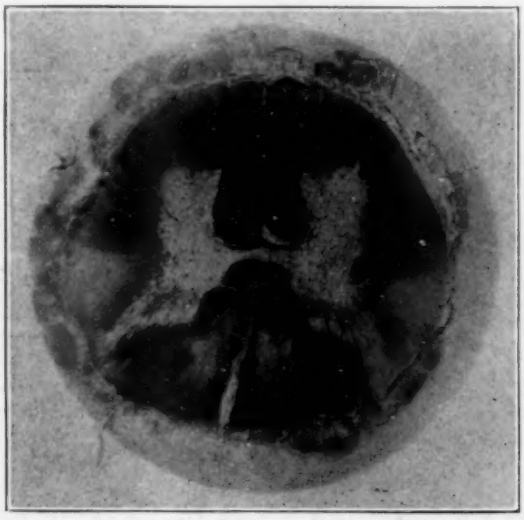

Fig. 12 (Case 8).—First lumbar segment cord; Weigert stain.

Putnam and Taylor [23] studied pathologically five cases, in all of which the patients had distinct sensory disturbances. In all there were relatively insignificant changes in the posterior roots, which the authors interpreted as local nutritive disturbances. Brown, Langdon and Wolfstein [42] stated that "probably Lissauer's zone is intact. In the dorsal and cervical levels the entering posterior root bundles appear slightly involved." Homen [43] found the posterior roots usually free though slight changes may appear with the Marchi stain. In the peripheral nerves few or no changes were found.

42. Brown, M. A.; Langdon, F. W., and Wolfstein, D. I.: Combined Sclerosis of the Lichtheim-Putnam-Dana Type Accompanying Pernicious Anemia, J. A. M. A. **36**:552-554, 1901.

43. Homen, A.: Des lésions non tabétiques des cordons postérieurs de la moëlle epinière, Rev. neurol. n. s. **8**:930-941, 1900.

Henneberg [44] (Lewandowsky) believed that the anterior and posterior roots are intact. The numerous black dots at the root entrance zone, exceptionally seen in Marchi preparations, are looked on as postmortem alterations. Bramwell [24] believed absence of lesions in the peripheral nerves to be characteristic.

In our own cases, we have tried to distinguish clearly between changes in proximal nerve trunks and in more distal nerves, but we have avoided small nerves of the extreme periphery on account of the difficulty in determining the presence of changes. As far as possible, we have drawn our conclusions from observations on the anterior tibial studied with the Weigert, Marchi and Bielschowsky methods. We believe that degeneration in the true peripheral nerves may be apparent

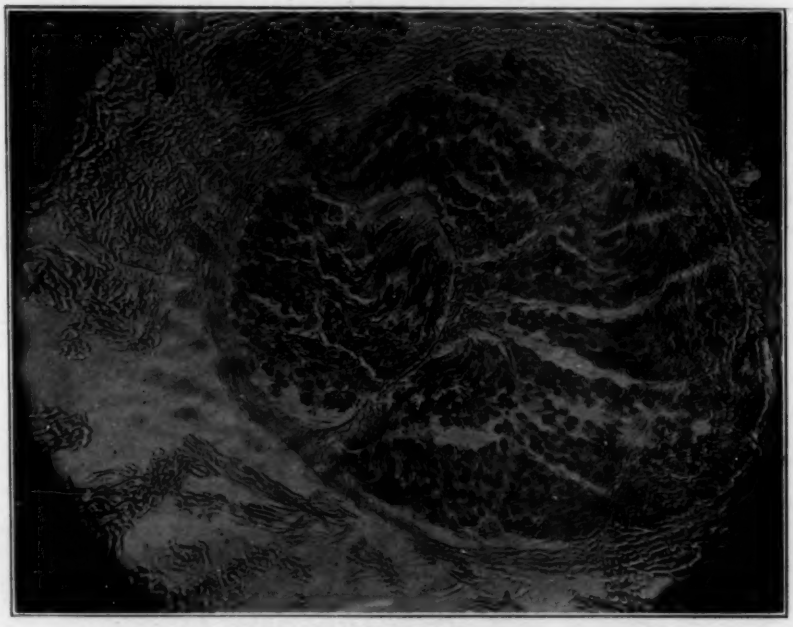

Fig. 13 (Case 8).—Peripheral nerve; Marchi stain.

with the Marchi method when Weigert preparations show nothing, and that the Marchi changes are not of agonal or postmortem origin. The peripheral nerves were studied in seven of the ten cases reported at some length. The Marchi method showed definite degeneration in Cases 1, 2, 3, 4, 8 and 9. The Weigert method gave similar results in Cases 2, 3, 4 and 6, and in Case 1 the nerves stained somewhat irregularly, which may be interpreted as possible evidence of beginning degen-

44. Lewandowsky: Handbuch der Neurologie, Berlin, Spezielle neurologie 2, **3**:99, 1910.

eration. A Marchi stain was not made in Case 6, and no peripheral nerves were obtained in Cases 5, 7 and 10.

In two of these cases Lissauer's tract was extensively involved; in two cases the root entrance zone was definitely involved on one or both sides, and in two other cases the degeneration was less distinct but fairly definitely shown by the Weigert method.

RELATION OF SPLENECTOMY AND OTHER FORMS OF TREATMENT AND OF REMISSIONS IN THE BLOOD PICTURE TO THE NEUROLOGIC PHENOMENA

Russell, Batten and Collier [20] note "the absence of any improvement of the nervous symptoms under treatment. From the first appearance of the disease to the fatal event, the tendency was for the symptoms to

Fig. 14 (Case 8).—Posterior roots; Marchi stain.

increase, sometimes halting, sometimes progressing rapidly, but never showing any remission." This is the experience of McPhedran,[30] Decastello [45] and Bloomfield except that the last author noted slight improvement in one case.

Three of our own patients had splenectomy performed. In one, following the operation, there was distinct advance in all the neurologic signs, including increase in the extent of the paresthesias and touch,

45. Decastello, v. A.: Perniciöse Anämie mit tabeformer Rückenmarksveränderungen und diffuse Sklerodermie, Ztschr. f. d. ges. Neurol. u. Psychiat. **5**:566, 1914.

joint and vibration blunting. The other two patients showed transient improvement both of blood and sensory disturbances.

Though sensory changes in our cases have usually been little altered during remissions in the blood condition we cannot agree that the nervous symptoms never improve. In one case, not reported in our present series, a physician showed marked sensory changes, both subjective and objective, for eighteen months before there was a distinctive blood picture of pernicious anemia. Subsequently he became so ill that he was confined to his bed for several months with loss of patellar and Achilles' reflexes and practically total loss of touch, pain, temperature, joint and vibration sensibility in the feet and legs and considerably less marked changes in the hands. Later his condition improved, and he

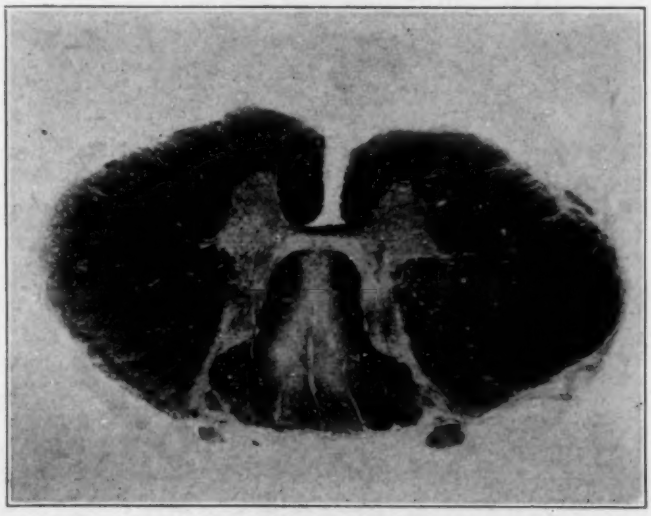

Fig. 15 (Case 9).—Second thoracic segment cord; Weigert stain.

returned to practice for a year. During this time his blood became almost normal, as did his appreciation of touch and pain stimuli with definite improvement in vibration sensibility and sense of position. After about twelve months the condition of the blood became much worse, his sensory disturbances reappeared, and he died within a short time. Though this is the most striking case in our experience of a remission in the sensory symptoms, in several other cases there was at times a distinct betterment in the course of the disease, capable of demonstration by objective methods. If our contention in reference to the occurrence of peripheral nerve changes be true, it is to be expected that there would be periods of improvement in the sensory findings in some cases.

PHYSIOLOGY OF SENSATION AND CORRELATION OF SENSORY AND ANATOMIC FINDINGS

As a basis for possible correlation of the anatomic and sensory findings, we briefly summarize the present views on the sensory tracts of the cord.

Two paths are open to touch: one through the uncrossed exogenous fibers of the posterior columns, another through the opposite anterior segment of the ascending anterolateral column. Impulses arising from painful and thermal stimuli enter the cord in short root fibers and are transmitted to the optic thalamus by fibers of the posterior segment of the ascending anterolateral column (tractus spinothalamic et tectalis).

Fibers for the sense of position and movement (Head and Thompson), vibration (Egger), stereognosis, tactile discrimination (compass

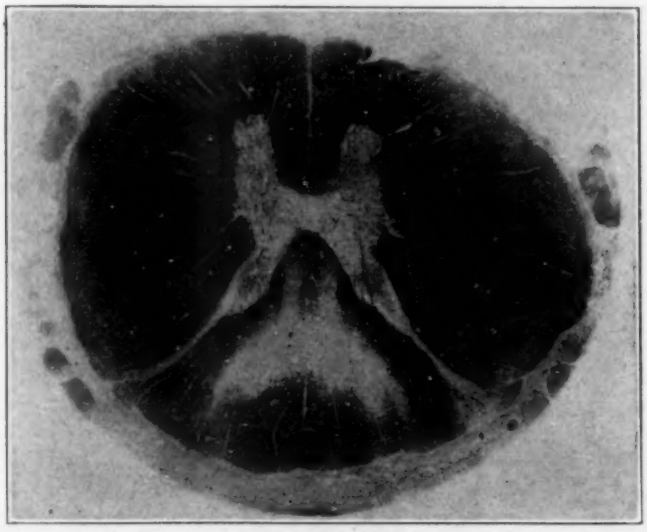

Fig. 16 (Case 9).—Seventh thoracic segment cord; Weigert stain.

test) and appreciation of weight (Head and Holmes, Déjerine, Page May, Sherrington) pass by way of the homolateral dorsal columns.

Deep unconscious sensibility (afferent impulses underlying muscle coordination and reflex muscular tone) is transmitted to the cord by short and median root fibers, receives collateral stimuli from long root fibers (Déjerine), and passes homolaterally in Gowers' tract and hereterolaterally in the direct spinocerebellar tract.

Déjerine states that "as to deep conscious sensibility (vibration, stereognostic and weight sensations, bone sensibility and tactile discrimination) it follows the course of the long root fibers of the posterior columns of the cord and enters its secondary pathway only at the level of the neck of the medulla."

Petren puts forth the theory that there is a double pathway in the cord for the sense of passive position; one in the posterior column, the other in the homolateral dorsal cerebellar tract. He believes that one of these pathways may be abolished without marked disturbance of passive position sense. From the clinical and pathologic study of a case, Thompson concludes that a complete lesion of the long fibers of the posterior column of the spinal cord results in a loss of the sense of passive position, ataxia, and loss of discrimination of points and weights. Localization of touch is little affected. This result agrees with the experiments of Page May. Painful, thermal, and tactile impulses can ascend the cord, although the posterior columns are degenerated. The evidence derived from Thompson's case is at variance

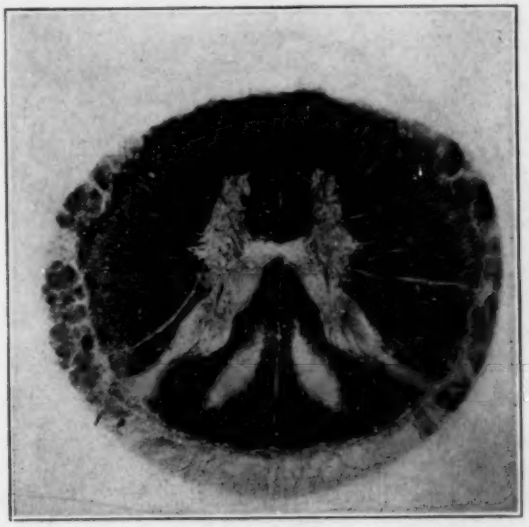

Fig. 17 (Case 9).—Twelfth thoracic segment cord; Weigert stain.

with the view of Petren that a lesion of the posterior column does not produce loss of passive position sense unless the dorsal spinocerebellar tract in the same half of the spinal cord is likewise affected.

A comparison of the objective sensory with the postmortem findings in ten selected cases of our series gives these results:

Case 1 (J. B.).—Vibration sense was impaired in the fingers and was absent in the feet and ankles. Position sense was normal. Sense of touch was diminished in the tips of the fingers. Superficial pain sense was normal. The cord showed a slight lateral change at the periphery and a slight change along the dorsal groove. There was some degeneration of the anterior and posterior roots and involvement of the peripheral nerves.

Case 2 (S. H.).—Vibration was impaired in the hands and absent in the feet and legs. Position sense was not noted. Sense of touch was diminished in the feet, legs and hands. Sense of pain was much impaired in the feet and

legs and less so in the hands. The cord showed marked degeneration in the posterior and lateral columns. Lissauer's zone was much involved, and there was extreme degeneration of the peripheral nerves (Figs. 1, 2, 3, 4).

Case 3 (M. P.).—Vibration sense was absent in the feet and legs. Touch, superficial pain and position sense were all normal. There was marked change in the posterior and lateral columns, the lesions in the posterior columns being mostly "patchy." There was marked degeneration, shown by the Marchi method, of the posterior and anterior roots and the peripheral nerves (Figs. 5, 6, 7, 8).

Case 4 (A. H.).—Vibration sense was absent in the feet. Sense of touch, pain and position were normal. The only change in the cord was a possible pallor along the dorsal groove. The peripheral nerves showed marked degeneration (Fig. 9).

Fig. 18 (Case 9).—Peripheral nerve; Weigert stain.

Case 5 (W. M.).—Vibration sense was not recorded. Position sense was absent in the toes. There was slight diminution of touch and pain sensation in the feet. There was marked change in the anterior, posterior and lateral columns of the cord. Some degeneration of the root entrance zone had occurred. The peripheral nerves were not available (Fig. 10).

Case 6 (A. R. S.).—Vibration sense was not tested. Position sense was absent in the feet and diminished in the hands. Touch and superficial pain sensation were slightly impaired in the feet and hands. The cord showed old sclerosis in Goll's column with patchy lesions in Burdach's column; there were both old and recent degeneration in the lateral columns and definite degeneration of the peripheral nerves.

Case 7 (H. W. L.).—Vibration sense was absent in the feet and legs. There was slight impairment of position sense in the toes. Anesthesia and analgesia of feet, hypesthesia and hypalgesia of the hands and legs were present. There was a sclerotic patch in the middle of one of the posterior columns; a slight

change had occurred in the other posterior column; there were practically no lateral changes. The peripheral nerves were not available (Fig. 11).

Case 8 (N. H.).—Vibration sense was absent below the midthoracic region and absent or much impaired in the hands. Position sense was absent in the toes. Sensation of touch was impaired in the feet. Pain sense was normal. There was degeneration in both lateral and posterior columns. The sciatic nerve showed some degeneration (Figs. 12, 13, 14).

Case 9 (H. S.).—Vibration sense was not recorded. Position sense was absent in the feet. There was slight hypesthesia in the toes. The cord showed a slight change in the lateral columns, at the extreme margin, with circumscribed sclerosis in the posterior columns. Degeneration was evident in the anterior and posterior roots and in the peripheral nerves (Figs. 15, 16, 17, 18, 19).

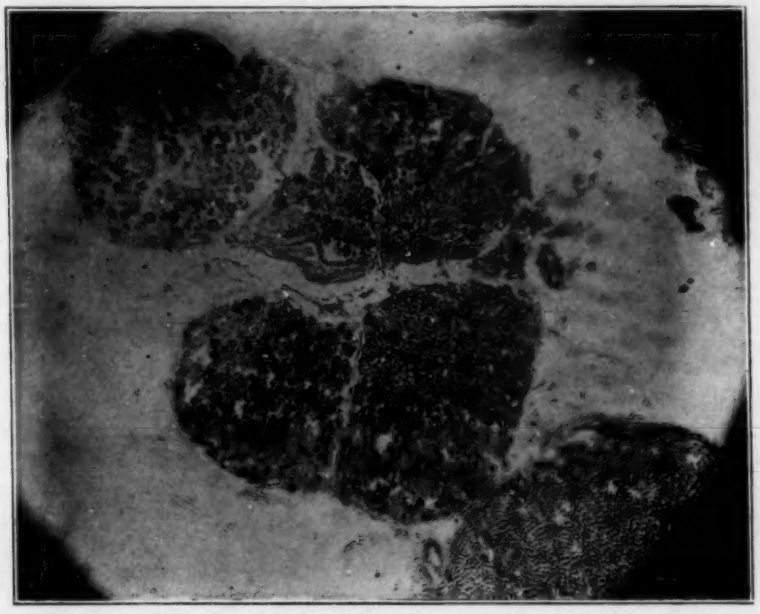

Fig. 19 (Case 9).—Posterior roots; Marchi stain.

Case 10 (A. P. M.).—Vibration sense was absent below the twelfth thoracic segment and much impaired in the hands and arms. Position sense was absent in the fingers, toes and feet. Touch and superficial pain sense were normal. There were a few ballooned fibers in the lateral columns and considerably more, rather diffusely scattered, in the posterior columns with an old sclerotic patch in the middle of each posterior column. The roots were uncertain. The peripheral nerves were not available.

Vibration sense was absent in seven of these cases, and three times it was not recorded. Of these seven, the posterior columns showed slight sclerosis in three, marked sclerosis in three and practically none in one. Of those showing slight sclerosis in the posterior columns, the lateral columns were normal in one and slightly involved in two. In the three patients with marked posterior sclerosis, the lateral columns

were similarly affected. The cord with practically no lesions in the posterior columns showed no change in the lateral columns.

In each instance except one, in which vibration sense was impaired or lost, the changes in the cord afforded a more or less adequate explanation in keeping with Déjerine's view. In this one instance a marked peripheral neuritis was present, subjective sensory phenomena were abundant and most unpleasant, and there was impairment of touch sensibility in the fingers, but pain and joint sensibility remained normal, and it is difficult to explain the extreme and selective loss of sense of vibration on the ground of peripheral nerve disturbances alone. In one instance (Case 7, Fig. 11) with marked loss of vibration in feet and legs there was a small clearly circumscribed patch in one posterior column, a less marked change in the other posterior column and practically no lateral change. It may be assumed in this case that the lesion in the posterior columns corresponds to the point of passage of the fibers conducting vibration sensibility.

Position sense was absent in five cases, impaired in one, normal in three and not recorded in one. In all of the five cases, there was marked degeneration of the posterior columns and in three of these the lateral columns were also much degenerated. In the remaining two the lateral columns were only slightly involved, and it would seem that, if joint sensibility were partly carried in the lateral columns, it would not have been wholly lost, as was true in these two cases. In the one case with impaired position sense, the posterior columns showed slight changes with slight lateral changes. Of the three cases in which position sense was intact, in one case the posterior columns showed numerous patches of sclerosis with more or less normal tissue between and marked change in the lateral columns (Figs. 5, 6, 7). The remaining two showed little change in the lateral columns and slight diffuse change in the posterior columns. In the one case, then, with impaired joint sensibility and in two of the three with normal sensibility the condition in the posterior and lateral columns was such as to indicate only a partial loss of function; these cases therefore do not throw any satisfactory light on the problem of the point of passage of the fibers carrying sense of passive position.

Superficial pain sense was absent in one patient who had a sclerotic patch in the middle of one posterior column and slight changes in the opposite posterior column and in the lateral columns. No peripheral nerves were available. Pain sense was markedly involved in one case with extensive sclerosis in the lateral and posterior columns. The two cases with slight hypalgesia of the feet both showed marked sclerosis of the lateral columns as well as of the posterior columns. In the four cases with no impairment of superficial pain sense the posterior and

lateral columns were normal in one instance. In a second instance there was slight change in the lateral columns with moderate involvement of the posterior columns and in the other two both posterior and lateral columns were more or less markedly degenerated.

Cotton touch was lost in one case showing only slight change in the lateral columns and in one posterior column and a small patch in the opposite posterior column. It was impaired in three cases of which one showed slight change in the lateral columns and along the dorsal groove and two showed marked degeneration in the lateral and dorsal columns. There was slight impairment in three cases, two of which had marked sclerosis in the lateral and posterior columns, and one had slight degeneration at the extreme lateral margin and a small patch in the posterior columns. Three cases had no involvement of touch sense, the cord being practically normal in one case, the lateral columns showing slight involvement and the posterior showing moderate sclerosis in the second, and in the third both lateral and posterior columns were extensively involved.

CLINICAL ANALYSIS

The following clinical analysis is based on the study of forty-one cases. As the completeness of the study varied in different instances, the tables do not always cover the full number of cases.

TABLE 1.—CLINICAL PHENOMENA

Age at Onset Years	Cases
26-30	1
31-35	3
36-40	6
41-45	7
46-50	11
51-55	4
56-60	2
61-65	4
66-70	2
71-75	1

Past Illness.—Under this heading nothing significant was ascertained.

TABLE 2.—DURATION OF ILLNESS

	Cases
6 months or less	3
7 months to 1 year	12
2 years or less	12
3 years or less	6
4 years or less	2
5 years or less	1
6 years or less	1

Early Symptoms.—The histories were taken by various interns, and although they may have erred in not obtaining all the early symptoms, they were at least free from any preliminary bias.

TABLE 3.—Early Symptoms

	Cases
General weakness	11
Paresthesia or numbness	12
Paresthesia or numbness and general weakness combined	14
Irritative signs, such as pain and soreness	8
Sharp pain	5

It has been our experience that many patients do not distinguish clearly between motor and sensory phenomena in using the term weakness or numbness and on this account the statistics appearing in the foregoing are not altogether reliable.

Complaint on Admission to Hospital.—This was more or less similar to the early symptoms.

TABLE 4.—Symptoms on Admission to Hospital

	Cases
Weakness	12
Paresthesia or numbness	3
Paresthesia or numbness and weakness	27
Irritative signs	10
Constipation	6
Urinary incontinence	6
Bowel incontinence	1
Shortness of breath	9
Pain in abdomen	7

Irritative phenomena differed. One patient complained of tenderness on pressure over both hands; another, of aching pains. One patient complained of pains in the lower extremities and of a sensation of cold in both upper and lower extremities, and one of rheumatism; one patient complained of continuous tinnitus over a period of two years and one of noises in the head. Another spoke of cramps and pains in the legs and feet.

Subjective Sensation.—The most common complaint was tingling and numbness. This was confined to the hands twice and the feet once, and was present in both hands and feet nineteen times. Pain was complained of in the hands and legs five times, in the arms and legs twice and in the arms alone not at all. A band sensation was described twice. Many times the patient found great difficulty in describing the sensation. One physician in particular, who was under observation for over three years, often referred to his sensations and would generally end by saying that it was a queer feeling which he could not accurately describe. One patient spoke of a feeling of "fine gravel in the hands and feet which she would like to pick off." Another spoke of "creepy, crawling feelings" and at times of a "sensation like sand." Another time she said her hands did not "feel right" and in further explanation added: "I can't describe that feeling—a week ago it was different, it was feverish and not stiff like now."

A third patient spoke of "a sort of roughness that came on as weakness." Another spoke of "a roughness—a bad feeling as if the muscles and nerves were wires, as if they were dry." A feeling like electricity passing through the body or hands and arms was often complained of. A sensation like needles so that it was painful to pick up objects was frequent. Some mentioned a wooden feeling in the fingers. One said his fingers felt as if they were made of glass, and that when he tried to pick up something he would be unable to hold it because he could not feel the object in his grasp. One man said it was difficult to tie his shoe strings because his "fingers did not seem to know where they were," and still another spoke of the difficulty or impossibility of buttoning his clothes because of the "sticking" feeling in his fingers.

A patient recently treated (W. G.) had had numbness in his hands and feet for twenty-two months. At the present time his fingers feel as if "they were crusted due to a burn with hot sand." When he touches anything with his fingers or toes a feeling of electricity seems to go through his body.

Objective Sensation.—Most of the sensory tests were made with a wisp of cotton, a pin with a sharp point and a spherical head and a tuning fork (256C). Deep pain sensibility and joint sensibility were also tested.

TABLE 5.—COTTON TOUCH

	Cases
Hypesthesia of hands and arms	6
Hypesthesia of feet and legs	4
Hypesthesia of arms and legs	10
Anesthesia of hands and arms	2
Anesthesia of feet and legs	6
Normal to cotton touch	10
Not tested	5

Hyperesthesia was present in four instances. Two of the patients suffered greatly from spontaneous pain, the other two did not. One patient showed hypesthesia from the second intercostal space to the lower costal margin. Sensation was normal in the abdomen and hypesthesia was present in the legs. Such diminution of sensation as was present varied from the slightest changes which were distinguished with great difficulty to those in which absolute anesthesia was present. These higher grades of diminution, however, did not go above the fingers or toes except in one instance in which there was practically no sensation up to the twelfth thoracic segment.

TABLE 6.—Pin Prick

	Cases
Hypalgesia of hands and arms	3
Hypalgesia of feet and legs	3
Hypalgesia of both	13
Analgesia of toes	1
Hyperalgesia of hands and feet	4
Hyperalgesia of varying degrees of fifth cranial nerve	7
No loss of sensibility to pin prick	10
Not carefully tested	9

Hypalgesia was present from the second intercostal space to the lower margin of the ribs in one case, as was true of the hypesthesia but, unlike the latter, it did not reappear in the legs. One patient spoke of a "burning sensation lasting several seconds" after being tested with the pin point.

Like hypesthesia, hypalgesia was usually present only in the fingers and toes, but in a fair number of instances it was present in the hands and feet as well, and in a few instances the patient would repeatedly miscall sharp and dull over any part of the extremities.

TABLE 7.—Vibration Sensibility

	Cases
Diminished in upper extremities	9
Diminished in lower extremities	1
Diminished in both upper and lower extremities	2
Absent in upper extremities only	0
Absent in lower extremities only	20
Absent in both upper and lower extremities	3
Normal in both upper and lower extremities	5
Not examined	8

Unlike the sensibility to touch and pain, vibration sensibility, when impaired, was greatly diminished or lost in practically every case over wide areas. In one instance vibration sensibility was found present throughout several examinations in a man who had been ill ten months. Subjectively, he complained of pain in the lower part of his legs and of numbness extending to the elbows and hips. He remained at the hospital nine months, and in the course of this time his subjective sensory trouble became distinctly worse. In all his examinations there were definite impairment of touch sensibility and slight impairment of pin prick. Position sense was present in the fingers and toes at first, but two months after admission it was lost in the toes.

As our table shows, vibration sensibility was very much more impaired in the lower than in the upper extremities. It was wholly absent twenty times in the lower extremities only, three times in both upper and lower extremities, and in no case in the upper extremities alone. How far upward the loss of vibration sensibility extended we are unable to say with any degree of accuracy. In most of our cases it was absent as far up as the malleoli and many times over the tibia, the

patella and even higher up. It is worthy of note that in some cases vibration sensibility was absent in one malleolus and present in the other, as Minnich found in one case in reference to superficial pain sense. Vibration sense was lost, therefore, in the upper or lower extremities or in both in twenty-three cases out of forty-one.

TABLE 8.—POSITION SENSE

	Cases
Sense of position absent in toes	9
Sense of position impaired in toes	9
Sense of position normal in toes	11
Sense of position not tested	12

Judged by our statistics, impairment of the sense of position is by no means so common as loss of vibration sensibility and is hardly more common in our records than diminution or loss of sensibility for touch or pain. We believe that loss of sense of position is less common in the fingers than in the toes. In one case (C) both vibration sense and sense of position were greatly impaired in the toes, whereas both were normal in the fingers. It is rather notable in this case that subjective sensory troubles were confined to the lower extremities. In a second case sense of position was normal in the fingers and slightly impaired in the toes. Vibration sense was absent in the toes, but not noted in the fingers. In this case the sensory trouble appeared in the hands previous to its appearance in the feet. In a third case sense of position was lost in the toes in the course of our observation of the patient, though the vibration sense remained at all times present. This patient had definite subjective sensory trouble in both hands and feet and a hypesthesia and hypalgesia of both.

Astereognosis was noted in three of our cases, but the study was not pursued sufficiently far to enable us to draw any conclusions.

Deep muscle pain sensibility was not carefully studied, but in thirteen instances it was noted as normal in the calves, three times increased in the calves and twice impaired, and twenty-three times it was not examined.

A CLINICAL ANALYSIS OF TEN CASES

We have selected ten cases from our series, in which the neurologic examination was more or less complete, for the study of the relative frequency of the sensory findings. These cases were chosen on account of the completeness of the study and not because of the presence of neurologic findings, though it is probable that the presence of many neurologic phenomena would lead to the more complete study of any given case.

Subjective Sensory Disturbances.—Paresthesias were present in all these cases—in eight they occurred early, either alone or combined with

weakness or gastro-intestinal disturbances. With few exceptions, the subjective sensory disturbances were present constantly and in greater or lesser degree.

Objective Sensory Phenomena.—Vibration sense was impaired or absent in the lower extremities alone three times and in both arms and legs five times; there was no impairment in two cases.

Position sense was lost in four of these ten cases; it was impaired in two; slightly diminished in one, and normal in three. It was absent or much impaired in four cases in which vibration sense was also absent, but was normal in three cases and slightly impaired in one case in which vibration sense was lost.

Touch, as tested with cotton, was normal in two cases, slightly impaired in two, and greatly diminished or absent in five cases. Twice there was hyperesthesia.

Superficial pain sense, as tested with a pin, showed no involvement in four patients, slight diminution in one and was much impaired or absent in four cases. In one instance there was definite hyperalgesia.

To summarize: One hundred per cent. of the patients had subjective sensory disturbances, 80 per cent. showed loss of vibration sense over several or more bony prominences, and 70 per cent. showed definite involvement of position sense in the toes. Touch sense was diminished or absent in some areas in 70 per cent. and superficial pain sense in 50 per cent. of the patients.

In one case in which vibration sense was later absent in both feet and hands, it was present during the first part of the patient's stay in the hospital at a time when position sense was completely lost in the toes.

CONCLUSIONS

Changes in the nervous system occur in pernicious anemia in 75 to 80 per cent. of the patients as they present themselves in the ordinary physician's practice.

The frequency of these changes is ordinarily underestimated, due to lack of care in the neurologic examination, or even to lack of any neurologic examination.

Subjective sensory disturbances constitute the earliest and most frequent evidence of involvement of the nervous system.

These, with the objective signs also, are often the earliest evidence of the development of pernicious anemia.

The most characteristic sensory findings are the relatively greater loss of deep as compared with superficial sensibility, but there is by no means normal sensibility to touch, prick and other forms of superficial sensibility.

Almost equally as characteristic as the change in deep sensibility, is the complaint of subjective sensory disturbances. Their constancy, their steady progress and symmetrical location in stocking or glove distributions, the inability of the patient to describe them in definite terms and the great distress without ordinarily actual pain to which they give rise, is rarely if ever found in any other clinical condition.

There is a distinct lack of parallelism between the severity of the blood and nervous phenomena, and either may precede the other by a long period of time. Moreover, marked remissions in the blood picture, either with or without splenectomy, may fail to show a betterment in the sensory disturbances equal to that in the blood, and may show none at all.

A similar disharmony may exist between the sensory changes and the postmortem findings in the cord, so that an essentially normal cord may be found in a person who had shown marked sensory disturbances both subjective and objective.

Contrary to the generally expressed opinion, degenerative changes in the peripheral nerves are common in pernicious anemia and constitute an important part of the pathologic anatomy of that disease.

These changes in the peripheral nerves may serve to explain the disharmony between the sensory phenomena and the changes in the spinal cord existing in certain cases, and in all cases have to do with the production of sensory disturbances; they are especially important in those cases in which marked remissions of sensory phenomena occur.

Vibration sensibility may be lost without loss of sense of passive joint movement and vice versa, and it is possible that the long fibers carrying these two forms of sensibility pass up the cord sufficiently separated so that a beginning lesion in the posterior column may involve either one or the other alone at an early stage or, in accordance with Petren's view, it may be that the fibers subserving joint sensibility, at least, may pass up the cord in two pathways.

MYELITIS AND MYELOMALACIA

A CLINICOPATHOLOGIC STUDY WITH REMARKS ON THE FATE OF GITTER CELLS *

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The term myelitis generally covers a great variety of spinal cord lesions, such as inflammations, degenerations and necrobiotic changes, caused by thrombosis, embolism and endarteritis. The clinical picture may be identical, and a proper clinical diagnosis of the actual condition, whether inflammatory, or degenerative, cannot always be made. In one of the cases recorded here the morbid phenomena were most probably due to a pure inflammatory condition of the spinal cord, myelitis, while the second case with the clinical picture of transverse myelitis was due to an extensive softening of the spinal cord with interesting histologic features.

CLINICAL REPORT (DR. BASSOE)

CASE 1.—*History.*—A Chinese laborer, 50 years of age, unmarried, was admitted to the surgical service of Dr. Carl B. Davis in the Presbyterian hospital, April 1, 1920, on account of urinary retention and weakness of the arms and legs. It was quickly determined that no surgical condition was present, and he was transferred to the neurologic service. Two weeks previously he had developed a cough, and the right leg grew weak but was not painful. The other leg and both arms also grew weaker, and he became unable to pass urine. The previous history, as far as it could be ascertained, was negative except for an attack of gonorrhea a year before, but he denied chancre.

Examination.—On admittance the bladder was distended and catheterization was necessary. The pupils reacted normally but the right one was a little larger than the left. He could not walk but in bed was able to move his extremities in a very ataxic manner. The grip of the left hand was much weaker than that of the right. The knee and ankle reflexes were increased and ankle clonus obtained.

Laboratory Tests on Admittance.—The Wassermann reaction of the blood was negative, and of the spinal fluid weakly positive. The cell count was 16. The Ross-Jones globulin test was positive; Lange's gold test: 0122110000.

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* From the pathology laboratories of Cook County Hospital, Chicago, and Illinois State Psychopathic Institute. A brief report of these cases was made to the Chicago Neurological Society, Nov. 18, 1920.

The leukocyte count was 8,100; blood pressure—systolic, 104; diastolic, 66. The urine was negative, and no cystitis developed; catheterization was not necessary after April 5. There was no fever.

Course of Disease.—April 10: Flexion and extension of the right ankle were fairly strong. Dorsal extension of the left ankle was weak. Flexion and extension of the knees were quite strong. Flexion of the right hip was stronger than that of the left. Heel to knee test showed marked ataxia. The weakness and ataxia of the trunk muscles were relatively more marked than in the legs. The grip of the right hand was fair, of the left weak. The left wrist movement was weaker than the right. The patient could hold his arm up. The flexion of the elbows was relatively strong; extension was weaker, especially on the left side. There was moderate ataxia of the hands. There was stiffness of the neck or cranial nerve palsies; the pupils were equal. Bilateral ankle clonus and probably a bilateral Babinski sign were present. Knee reflexes were increased with prolonged patellar clonus on the left. The cremasteric reflexes were obtained, as well as the right abdominal, which was weak; the left was not obtained. Both wrist reflexes were present; the right elbow reflex was present, the left was absent. There was considerable rigidity of the trunk muscles, slight of the left leg, none of the right leg and arm muscles. Sensation apparently was normal; no tremor. Mentality was good.

April 16 the patient could raise himself half way to a sitting posture by using the arms, but with the hands folded on the chest could raise only the head. Abdominal reflexes were as before. Patellar clonus was not obtained; ankle clonus was present but less prolonged; the planter reflexes were doubtful.

Laboratory Tests on April 14.—The Wassermann reaction of the blood and spinal fluid was negative. The cell count was 9. The Ross-Jones test was weakly positive; Lange gold test: 0122211000.

Course of Disease.—May 7: The left grip was still rather weak. All finger movements, including opposition of thumb to little finger, could be made. The trunk muscles were weak, as before. Dorsal flexion and external and internal rotation of the left foot were apparently nil; of the right foot good. Flexion of the left knee was weak; of the right, fair. Extension of both knees was good. Flexion of the left hip was weak; other hip movements were good. Bilateral ankle clonus and slight left patellar clonus were present. Abdominal reflexes were absent.

The condition gradually improved and on July 25, the following note was made: "The patient walked today without a cane. The left leg is still weak, particularly as to eversion of the foot. Left ankle clonus and increased knee jerk persist. The left grip is stronger, and all movements of both wrists are strong.

The patient left the hospital on August 26 and when seen again, on October 15, only little weakness remained.

Comment.—When this case was reported to the Chicago Neurological Society I suggested it might have been one of infection with the virus of epidemic encephalitis, the cord being chiefly affected. However, the absence of cranial nerve symptoms, except for a transient inequality of the pupils and the early retention of the urine, make it more probable that we were dealing with a case of disseminated myelitis of different etiology.

Case 2.—*History.*—The patient was a married woman, 45 years old, of good family history and with good previous health. She had three healthy children,

and there was no reason to suspect syphilitic infection. The husband gave a negative Wassermann reaction. The patient was first seen by me, Sept. 22, 1919. Seven weeks previously she had complained of sore throat and rather severe nosebleed. The legs then became stiff, and after a couple of weeks, she complained of pain in the calves and thighs which was severe at times; she had not been able to walk for about three weeks at the time of my visit. The trouble with the legs was followed by numbness and weakness of the hands and frequent, precipitate micturition. Two weeks previously she had complained of diplopia which lasted for a few days only. She became somewhat dull mentally, and her speech was a little indistinct. There was no headache. She was so clumsy that she had to be fed. Occasional involuntary micturition and defecation had occurred.

Examination.—The patient was well nourished, very sluggish mentally, disoriented as to time and place, with slurred, thick speech. The pupils were equal; the left one reacted very sluggishly to light and in accommodation; the right one reacted better. There was nystagmus on looking to the right. There was no definite cranial nerve paralysis. The tongue was heavily coated, not tremulous. The neck was slightly stiff. There was no paralysis of the arms but coarse, irregular movements of the fingers and arms. Marked ataxia was shown on finger to finger and finger to nose tests. There was complete paralysis of both lower extremities. Wrist and elbow reflexes were normal. Abdominal reflexes were not obtained. Knee reflexes were moderately increased. Bilateral ankle clonus and Babinski sign were obtained. The legs were rather flaccid. Touch and pain senses were lost in both legs almost to the hips, but the upper border was very vague. There was complete loss of sense of passive movement in the toes.

Laboratory Tests.—The Wassermann test of the blood was negative, of the spinal fluid, weakly positive. The cell count was 26. The Ross-Jones globulin test was weakly positive; the Lange gold test rather strongly so (4333211100). There was 80 per cent. of hemoglobin. The red cells numbered 4,240,000; the white cells 12,100. The blood pressure was: systolic, 140; diastolic, 68. The urine was cloudy and contained a moderate number of leukocytes. The temperature was 98, the pulse rate 80.

Course of Disease.—September 24: There was more mental confusion. Left ankle clonus was obtained and the right ankle reflex was slightly increased. The knee jerks were increased without clonus.

September 26: The legs were entirely flaccid. Knee and ankle reflexes were absent. The patient complained of pain and soreness across the chest and back in the midthoracic region.

September 28: Considerable stiffness of the neck and apparent difficulty in turning the eyes to the left were present.

September 29: The leukocytes numbered 9,300. Apparently there was complete loss of motion and sensation below the level of the fifth rib. The plantar reflexes were absent. There was still difficulty in getting the patient to move either eye or both eyes together to the left.

October 3: Lumbar puncture was performed. The Wassermann reaction of the spinal fluid was weakly positive. The cell count was 37. The Ross-Jones test was weakly positive. Lange gold test: 1223211000.

October 8: The leukocytes numbered 16,900. There was constant urinary retention. Bedsores were developing rapidly. The temperature, which was normal on the day of admission, rose to 100 F., Sept. 27 and to 101 F., Oct. 11. From that time on the temperature was usually normal in the morning and up to 101 and sometimes 102 F. in the evening.

October 10: There was increasing difficulty in understanding. When told to touch the nose with the left thumb, she fumbled with the right hand. There was a tendency to euphoria.

October 14: Dr. Phemister made deep incisions through the necrotic area over the sacrum without using an anesthetic, but the patient felt nothing. The hands were more ataxic.

November 7: She was asleep most of the day but was restless at night. Her tongue was coated. She did not have a headache or other pain. She had urinary retention as before. There was no nystagmus except some coarse jerkings on looking to the right. The disks were normal with the exception of a suggestion of temporal pallor in the right eye. There were no cranial nerve palsies. Sensation was lost below the level of the fourth rib.

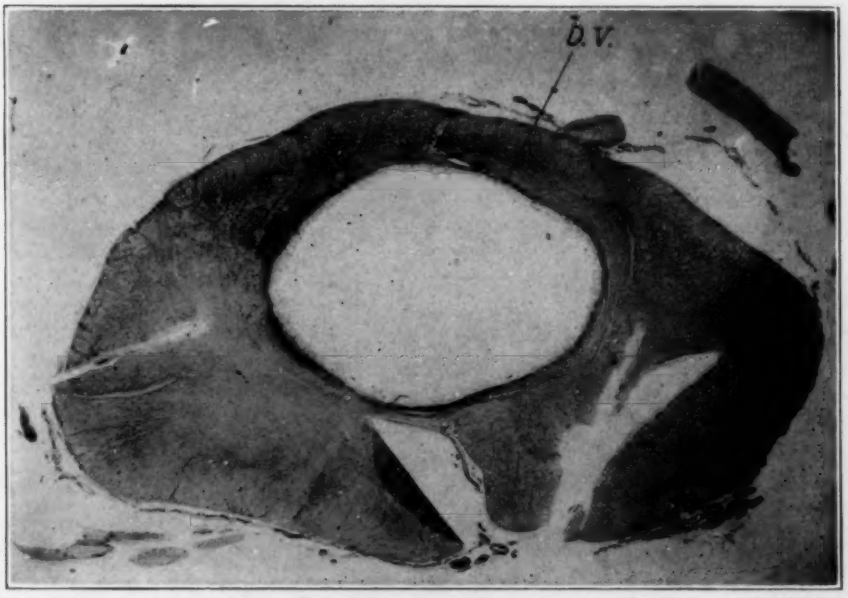

Fig. 1.—Cavity (dorsal region of the spinal cord) lined by a row of gitter cells which in their turn are surrounded by a visible membrane of thickened glia tissue and blood vessels. Alzheimer-Mann stain; × 8.

November 16: Lumbar puncture was performed, which was not felt by the patient. The fluid was clear, the pressure low. The Wassermann reaction of the blood and spinal fluid was negative. The cell count was 19. The Ross-Jones test was weakly positive; the Lange gold test 1222211000.

November 21: The pupils reacted well to light.

December 13: Extensive sloughs were excised from the bedsores over both trochanters.

December 21: The leukocytes numbered 13,600.

During the following month, the irregular fever persisted, the temperature at times reaching 104 F. Paraplegia, anesthesia, retention, bedsores and mental confusion remained as the principal symptoms. The patient gradually failed, and died Jan. 25, 1920.

PATHOLOGIC REPORT (DR. HASSIN)

Macroscopic Changes.—Practically absent in the cerebrum, they were striking in the spinal cord. On section, the entire dorsal portion of the latter was represented by a semiliquid, yellowish, creamy mass escaping freely on the slightest pressure on the membranes, leaving a distinct cavity (Fig. 1). Remnants of anterior and lateral columns could be made out in the upper and middle dorsal regions, while the lower dorsal portion was totally destroyed. Somewhat less damaged was the lumbosacral region in which only the posterior columns were involved. The cervical and lower sacral regions showed only secondary degeneration.

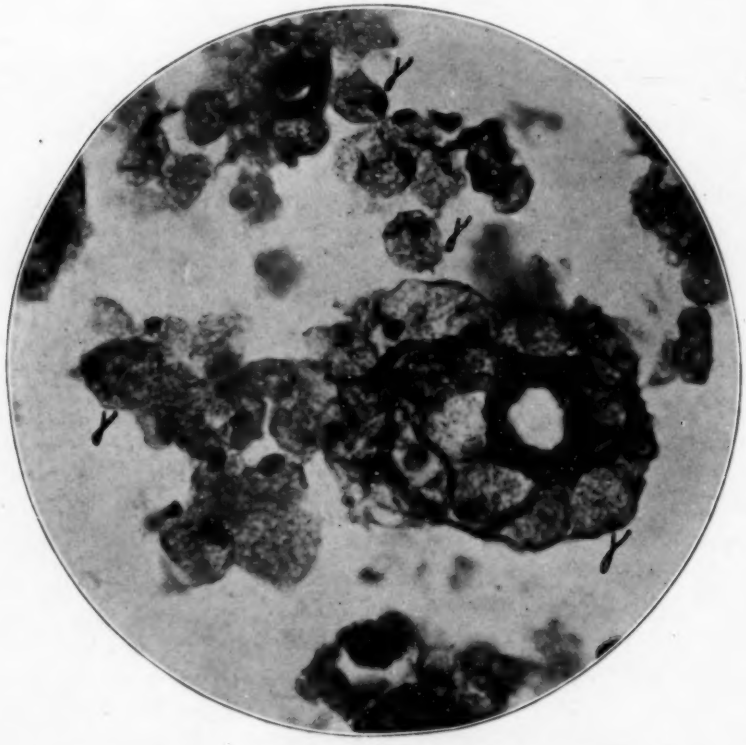

Fig. 2.—Gitter cells γ lying freely and around a small empty vessel enveloped by the ramifications of its thickened adventitia. The freely lying gitter cells show no capsule. Bielschowsky counterstained with Weigert's resorcin fuchsin; × 520.

Microscopic Examination.—Portions of the spinal cord and brain hardened in 10 per cent. formaldehyd have been studied in paraffin and celloidin sections. The dorsal portion consisted mostly of a mass of gitter cells scattered among vessels, broken up nerve fibers and numerous so-called islands of sclerosis. Occasionally one could find preserved ganglion cells. Some segments of the spinal cord showed small patches of gray matter containing practically normal ganglion cells but surrounded by a degenerated, broken up mass of white substance. The remaining gray substance of the thoracic portion appeared his-

tologically normal. Equally normal were the ganglion cells in the less damaged portions of the spinal cord, as well as in the medulla, midbrain, cortical areas and basal ganglions, except the corpus striatum which showed excessive and pronounced neurophagic phenomena.

The better preserved areas of the thoracic region exhibited an abundance of glia nuclei without marked increase in the amount of their cytoplasm. The lumbar region showed numerous gliogenous formations consisting of gitter cells which, in some areas, almost totally replaced the gray substance. The latter could be differentiated from the similarly changed white matter by the occasional presence of practically normal ganglion cells.

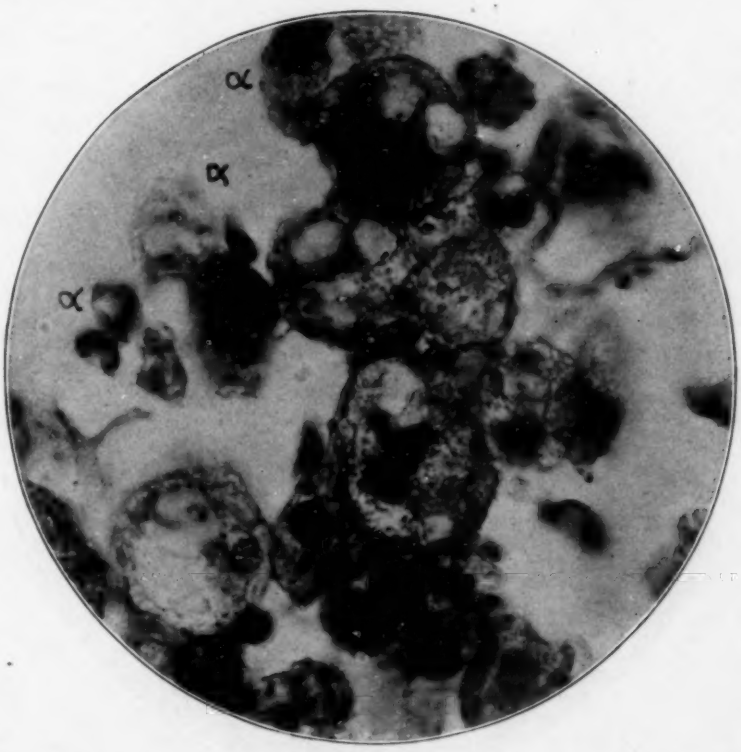

Fig. 3.—Groups of myelophages, large round bodies containing two, three and more large vacuoles with remnants (dark irregular bodies) of nerve tissue within. At α, gitter cells with an eccentric nucleus and a distinct vacuole. Bielschowsky counterstained with resorcin-fuchsin; × 1200.

The blood vessels of the gray matter were numerous, hyperemic, with thickened walls, but without perivascular infiltrations. A few hemorrhages were observed, but without any reactive phenomena on the part of the glia or mesodermic elements. They most likely were terminal manifestations.

White Substance.—Thinly spread between two slides and stained, some with a 1 per cent. solution of methyl-blue-eosin (Mann's mixture), others with osmic acid, scarlet red and Nile blue sulphate, the creamy mass showed an enormous number of various gliogenous formations, principally myelophages and gitter cells. Most numerous were gitter cells of Jacob's γ-variety, in the

form of round cell bodies, devoid of processes as well as of a visible membrane, with an eccentric nucleus and a distinct reticular or latticelike structure (Fig. 2). Invariably they were packed with small globules of fat. Certain cells appeared coarsely vacuolated (Fig. 3), with remnants of nerve tissue within some of the vacuoles. They are best classified as myelophages which are phagocytic cells. They usually contain fragments of myelin, which is transformed by them into fat. According to Jacob, the fat is deposited in gliogenous formations generally termed gitter cells. Such cells, of the γ-variety, were freely scattered in the tissues or gathered around the blood vessels, preferably the smaller ones, in the form of concentric rings (Fig. 2). The rings of γ-cells were separated from each other by connective tissue strands, with pro-

Fig. 4.—Islands of sclerosis, scl; hyperemic small veins, v; the rest of the field is covered with gitter cells and broken up nerve fibers. Paraffin section of an emulsified (softened) area. Bielschowsky stain; × 275.

longations surrounding each individual gitter cell. The latter thus appeared as if invested by a connective tissue membrane, a condition never seen in the ordinary gitter cells lying free. These membranes, as well as the strands of connective tissue dividing the rings of the gitter cells, were seen to be connected with the adventitia of the vessel wall.

Striking also was the presence around some capillaries of gitter cells still lying free but ready, so to speak, to be enveloped (Fig. 2) by the proliferating adventitia. It is to be noted that only the so-called γ-variety of gitter cells could be found near or within the adventitia, for only such gitter cells are

ready to discharge their fat. Myelophages, gitter cells of other varieties, such as those possessing vacuoles or processes, were not to be found within the adventitia.

Aside from the vessels surrounded by rings of gitter cells, there were a great many vessels densely surrounded, muff-like, by connective tissue which, however, showed no gitter cells in its meshes (Fig. 4). Such vessels had a barely visible lumen, but an enormously thickened wall. In fact, they appeared as islands of connective tissue or sclerosis, greatly resembling the islands in syringomyelia.[1] One gains the impression that they originate from the vessels surrounded by the gitter cells as well as from the connective tissue rings which,

Fig. 5.—The cerebellar pia. Excessive vascularization, hyperplasia and cellular infiltration of the distended meshes; *c*, cerebellum. Van Gieson stain; × 80.

after their contents, the gitter cells, have been removed, ultimately shrink forming islands of sclerosis. In many places, the field is represented merely by rows of pale gitter cells surrounding an empty space, or cavity. The rows of gitter cells lining the cavity are remnants of a great mass of similar structures that must have filled the space before the softening had reached the stage of cavity formation. The large cavity shown in Figure 1 was surrounded by a capsule rich in protoplasmic glia cells, blood vessels and glia fibers. Resem-

1. Hassin, G. B.: A Contribution to the Histopathology and Histogenesis of Syringomyelia, Arch. of Neurol. & Psychiat. **3**:130 (Feb.) 1920.

bling, at first glance, a similar formation commonly seen in syringomyelia, the described cavity is histologically and etiologically entirely different from that of syringomyelia.[1]

Scattered among vessels, vascular islands of sclerosis and gitter cells were nerve fibers, naked axons and fragments of myelin (Fig. 4). The nerve fibers were often practically unchanged, merely broken up into more or less well stained fragments, while some showed an enormously swollen or tumefied myelin sheath which was usually broken up into smaller fragments enclosed within myelophages and gitter cells. In areas that escaped softening, such as Gowers' tract, Flechsig's tract and the anterior colums, the nerve fibers were practically normal. The glia also was preserved, exhibiting an abundance of glia nuclei and so-called protoplasmic glia cells. The latter were often very large, in some instances resembling monster glia cells, and by their homogeneous staining, plump processes and eccentric nuclei, recalling at first glance so-called ameboid glia cells. However, if stained with Alzheimer-Mann mixture, Bielschowsky's method or even toluidin blue, they appeared exceedingly rich in chromatin nuclei and displayed a great wealth of ramifying protoplasmic processes. Nor could we find the "Füllkörperchen" of Alzheimer, or the "Abbau" products so abundantly present in some toxic-infectious conditions. In short, signs of regressive glia changes were totally absent. On the contrary, the glia exhibited marked progressive or proliferative phenomena, not only in the better preserved portions of the spinal cord, but also in those profoundly changed.

The vessels, as already noted, while exceedingly numerous in the destroyed area, mostly in the form of small capillaries with patent lumen and thickened adventitia, were practically normal in the preserved regions of the cord, as well as in the rest of the central nervous system. Only the lower medulla and the region of the brachium pontis exhibited vascular changes. In the medulla, some vessels were thrombosed and surrounded by an area of sclerosed tissue; in the pons there was a quite pronounced perivascular infiltration with plasma cells and lymphocytes.

The pia appeared hyperplastic, thickened and frequently packed with gitter-cells. The pia-arachnoid of the anterior longitudinal sulcus exhibited a great wealth of distended vessels, as well as various hematogenous elements, such as erythrocytes, leukocytes and plasma cells, while at some levels the subarachnoid space contained hemorrhagic foci. At other levels, instead of hemorrhages there were many gitter cells, mostly gathered within the meshes of the pia, around the vessels and, what is rather striking, in the posterior roots themselves.

Equally noteworthy were the findings in the cerebral subarachnoid space, especially at the base, around the optic chiasm, cerebellum and the occipital lobe. Figure 5 shows the pia-arachnoid around the cerebellum, with wide meshes, an enormous amount of blood vessels and various infiltration elements, such as lymphocytes, fibroblasts, clusters of mesothelial cells, gitter cells and hyperplastic strands of the pia-arachnoid. In other cortical regions such infiltrations were less in evidence, but hemorrhagic foci of various dimensions were quite common. However, as in the case of the spinal cord, the hemorrhagic foci were devoid of reactive phenomena and should therefore be considered as terminal phenomena. The choroid plexus was greatly vascularized and showed an abundance of connective tissue. The cells invariably showed the presence of fat in specimens stained with scarlet red and otherwise appeared unusually large and vacuolated.

Summary of Findings.—The findings were:

1. Absence of any inflammatory phenomena in the spinal cord or any other portion of the central nervous system, except some areas of the pons.
2. Vast degeneration of the spinal cord, especially the posterior portion, including some of the posterior roots.
3. An abundance of various gliogenous formations, such as gitter cells and myelophages, which almost totally replaced the degenerated areas.
4. Marked infiltration of distended adventitial meshes, pia and posterior roots with gitter cells and abundance of islands of sclerosis.
5. Hemorrhages in the subarachnoid space of the cervical region of the spinal cord.
6. Cellular infiltration of the distended pial meshes of the brain, especially around the base.
7. Fat infiltration of the choroid plexus.
8. Absence of regressive changes in the glia, in the presence of profound parenchymatous lesions.

DISCUSSION

The histopathologic findings here outlined in a typical case of so-called "yellow" softening of the spinal cord, myelomalacia, are analogous to those found in acute softening of the brain, encephalomalacia. As the latter is no longer considered an inflammatory process, neither can myelomalacia be looked on as a myelitis. Yet, inflammatory conditions ultimately may cause the softening described, for instance, in poliomyelitis, Pott's disease,[2] syphilitic cord lesions, and other conditions.[3] However, the areas of softening in such cases are usually small and circumscribed, while in the case under discussion, they involved the posterior portion of almost the entire spinal cord. The same is practically true for spinal cord softening due to intoxications as experimentally so well proved and studied, among others, by Lotmar.[4]

By far the most frequent cause of softening is vascular thrombosis ("thrombotic softening of the spinal cord" or "spinal thrombosis" of Singer [3]). According to Mager,[5] 80 per cent. of cases of so-called acute myelitis are due to necrobiotic or degenerative processes, usually brought on by vascular changes (infiltrative or proliferative). In the present case such changes were lacking. That the dorsal region was principally affected is probably due to the fact that the blood supply of this region is generally inefficient, and thus more easily implicated. Whatever the actual mechanism or nature of the vascular disorder in

2. Hassin, G. B.: Histopathologic Changes in Five Cases of Myelitis, Med. Rec., 1916, October 7.

3. Singer, H. D.: The Pathology of So-called Acute Myelitis, Brain **25**:332, 1902.

4. Lotmar, Fritz: Beiträge zur Histologie der akuten Myelitis und Encephalitis, sowie verwandter Prozesse. Nissl-Alzheimer's Arbeiten **6**:245, 1913.

5. Mager, W.: Ueber Myelitis acuta, Obersteiner's Arbeiten aus dem Institut für Anatomie und Physiologie an der Wiener Universität **7**:1, 1900.

this case, it is beyond question that the changes of the spinal cord were similar to those found in secondary degeneration. A typical instance of the latter is represented in this case by the posterior columns of the cervical region. The greatest portion of the nerve fibers were destroyed and replaced by numerous gliogenous formations, such as protoplasmic glia cells, abundance of glia fibers, an enormous amount of gitter cells and glia nuclei. All these formations were kept together, preserving the shape of the posterior column, while in the region of the softened area, the latter was a shapeless mass. In short, the gitter cells harboring the nerve tissue transformed into lipoid substances evidently tend to desert the destroyed area, to become "absorbed." The question arises: How do they accomplish the process of absorption, or what is their ultimate fate? Schmaus,[6] Nissl,[7] Merzbacher,[8] Marchand,[9] Held[10] and Jacob[11] tried to solve this problem. For instance, Schmaus and Nissl thought that the fat-laden gitter cells reaching the vessels unload their fat in the adventitial cells. Jacob is more explicit. According to this investigator, the fat taken over by the mesodermic elements of the vessels is "rebuilt," the adventitial cells turning into gitter cells. Merzbacher believes that these cells are absorbed either through the process of undergoing regressive changes and perishing or by being carried away in toto. He could find them, somewhat modified in structure, in the pia which he calls "Ablagerungsort" (dumping grounds). They reach the pia, he thinks, either by way of the lymph spaces or through the transformed adventitial cells. Finally, he admits that he does not know how the landing of the gitter cells in the adventitial spaces is accomplished, as neither he himself nor any one else ever succeeded in following up the process of direct invasion of the lymph spaces.

As shown in part by Figures 2, 3 and 4, the great majority of gitter cells are either freely scattered over the parenchyma of the spinal cord (Fig. 2), or are enclosed within (1) the meshes of the proliferated adventitia of the vessels, (2) the pia, (3) the subarachnoid space, or (4) the posterior roots. Wherever located, they are similar in structure;

6. Schmaus, H.: Vorlesungen über die pathologische Anatomie des Rückenmarks, 1901.

7. Nissl, F.: Kritische Bemerkungen zu H. Schmaus: Vorlesungen über die pathologische Anatomie des Rückenmarks, Centralbl. fur Nervenheilk. u. Psychiat. **26**:88, 1903.

8. Merzbacher, L.: Untersuchungen über die Morphologie und Biologie der Abraumzellen im Zentralnervensystem, Nissl-Alzheimer's Arb. **3**:1, 1910.

9. Marchand, F.: Untersuchungen über die Herkunft der Körnchenzellen des Zentralnervensystems, Ziegler's Beitr. z. pathol. Anat. **45**:161, 1909.

10. Held, H.: Ueber die Neuroglia marginalis der menschlichen Grosshirnrinde, Monatschr. f. Psychiat. u. Neurol. **26**:336, 1909.

11. Jacob, A.: Ueber die feinere Histologie der sekundären Faserdegeneration in der weissen Substanz des Rückenmarks, Nissl-Alzheimer's Arb. **5**:1, 1912.

the gitter cells of the perivascular or adventitial spaces, for instance, being entirely similar to those present in the meshes of the pia. The sole difference, as noted in the foregoing, is that some gitter cells, especially those freely scattered, are devoid of a connective tissue membrane which is so well seen around the cells within the adventitial spaces. These membranes can be seen originating from the adventitia whose ramifications form numerous meshes harboring the fat laden gitter cells (Fig. 2). Once within the meshes of the adventitial membrane, the gitter cells are carried away by the tissue fluids toward the arachnoid space in the same manner as is the case in the brain.[12]

The gitter cells found in such great numbers underneath the pia are removed in a somewhat analogous manner. They are gradually enveloped by the proliferating pia which thus brings them within the domain of the subarachnoid space and the posterior roots. An analogous process on the part of the pia is to be seen in syringomyelia in which the débris of glia and other tissues are actively removed by this proliterating membrane. However, while in syringomyelia the process of removal of destroyed tissues is exclusively carried out by the pia, this is in myelomalacia the final stage in ridding the nervous system of waste elements. The preliminary work is done by the glia which first transforms the damaged nerve tissue into lipoid substances and carries them to the mesodermic tissues, vessels or pia. The latter, by active proliferation envelop these harmful elements, and finally eliminate them to the subarachnoid space, "the dumping ground" of the central nervous system.

CONCLUSIONS

1. Myelomalacia is not an inflammation, but a degeneration of the spinal cord.

2. The degenerative or regressive nerve fiber changes are associated with progressive glia phenomena, principally in the form of gitter cell formation.

3. The gitter cells in this as in any other type of secondary degeneration, are gliogenous formations. Their task is to transform destroyed nerve tissue into fat-like substances and to transport these substances to mesodermic structures, such as blood vessels and the pia.

4. Reaching these structures, they are actively taken up by the proliferating adventitial and pial elements.

5. From the adventitial and pial spaces they are carried to the subarachnoid space and thence to the blood for final elimination.

6. The subarachnoid space is to be looked on as the "dumping ground" for any waste accumulated in the central nervous system.

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12. Hassin, G. B.: Histopathology of Brain Abscess, Arch. Neurol. & Psychiat. **3**:616 (June) 1920.

EPIDEMIC ENCEPHALITIS

CLINICAL AND PATHOLOGIC STUDY OF TWENTY-FIVE CASES

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INTRODUCTORY

In a previous communication, H. E. Allen and I[1] reported four cases of lethargic encephalitis as seen in the early part of the epidemic, and reviewed the literature and cases reported up to that time. After a short interval, during which few cases were seen in our community, a sudden recrudescence occurred. During this recrudescence I had the opportunity to study twenty-five cases of the disease and also to make necropsy examinations of several. These cases I shall report.

GENERAL CONSIDERATIONS

Age and Sex.—The youngest patient was 16 years of age; the oldest 61. Nineteen of the twenty-five were between 25 and 40 years old. Eighteen were males and seven females.

Relation to Influenza.—Direct: Of the twenty-five patients observed, only two gave a history of influenza. It consisted of a mild attack six weeks and four months, respectively, prior to the encephalitis.

Indirect: A definite relation between epidemic influenza and epidemic encephalitis was observed. The great epidemic of influenza raged during the months of October and November, 1918. We recognized the first case of epidemic encephalitis in February, 1919. Between February and October, 1919, we saw four typical cases, but from that time to February, 1920, hardly any cases were seen in the community.

A recrudescence of the influenza epidemic occurred in January, 1920, and lasted until Feb. 20, 1920, when cases of encephalitis began to appear. They gradually increased in number and reached their height in the latter part of March, when most of the cases were seen. On one day—March 24—five patients with encephalitis were admitted to the Waterbury Hospital.

Mode of Onset and Symptoms.—Premonitory symptoms of lassitude and general malaise of several days' duration were complained of by

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1. Alexander, M. E., and Allen, H. E.: Lethargic Encephalitis, Arch. Neurol. & Psychiat. **3**:485 (May) 1920.

ten of the patients. The most frequent mode of onset (sixteen cases) was with neuritic pain. In the majority of instances it commenced rather abruptly. Jerking and twitching were frequent symptoms and were among the first complained of in eighteen of the cases. In five cases the early symptoms were suggestive of a mild meningitis: rigidity of the neck, headache, tâche cerebrale and a positive Kernig's sign.

FEVER: A temperature above 100 F. was found at one time or another in all cases observed. In eighteen cases it was 102 F. or above. It was highest in the cases associated with a great deal of twitching and neuritic pain and in those that showed signs of cortical involvement. With exacerbations the temperature generally rose

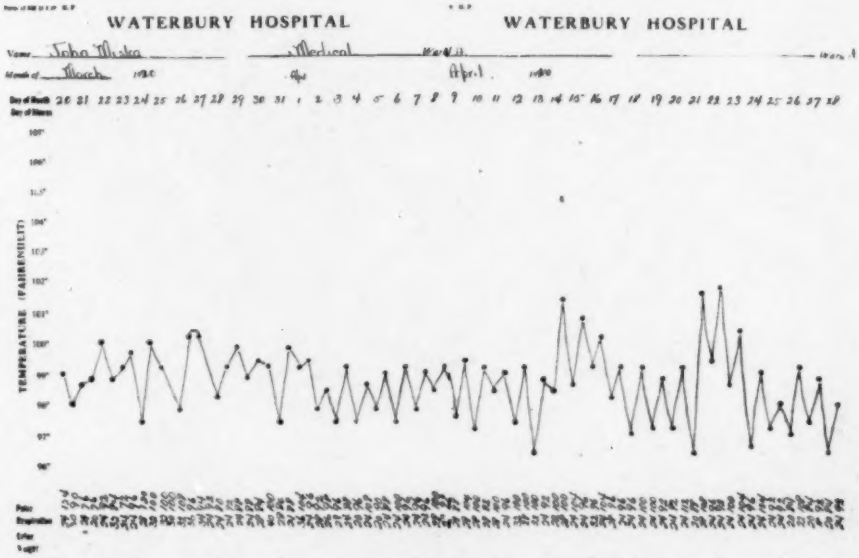


Fig. 1 (Case 10).—Usual temperature elevation in the meningoradicular type of encephalitis and the elevations with recrudescences of the disease.

(Fig. 1). In two cases hyperpyrexia was noted (Fig. 2). Both terminated fatally. The rise of temperature was irregular and unless taken at frequent intervals might have been overlooked in some cases.

PULSE: The pulse presented the usual features of infectious diseases. It averaged between 90 and 100 and was generally a little faster than one would expect with the comparatively slight elevations of temperature. In patients showing evidence of marked disturbance of the basal nuclei (dysphagia and marked lethargy), the pulse was generally between 100 and 110. A rate above 120 was uncommon, and when it persisted was of serious import. Extrasystole (transient) was noted in two cases; other irregularities of the pulse were absent.

BLOOD PRESSURE: This was generally slightly elevated, not only during the stage of excitement but even during pronounced lethargy. In one case peculiar, temporary elevations of blood pressure were observed. On admission to the hospital it was: systolic 120; diastolic, 70. On the second day it was systolic, 190; diastolic, 100. Similar fluctuations were noted on two other occasions.

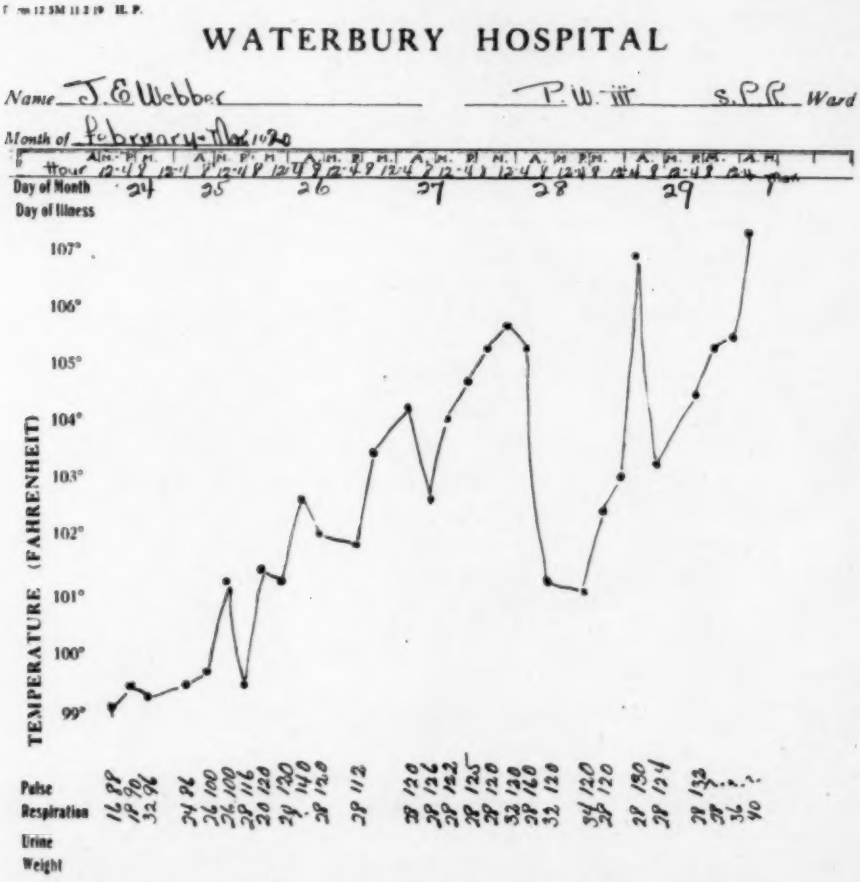


Fig. 2 (Case 16).—Temperature elevation showing hyperpyrexia. Necropsy examination revealed no complications and no pathologic findings except at the base of the brain.

CENTRAL NERVOUS SYSTEM

Restlessness and wakefulness were present in eighteen patients. In five, lethargy set in after a preliminary period of restlessness and insomnia lasting two or three weeks. These cases particularly emphasized the fact that the various cases which at certain times showed symptoms so vastly different, really belonged to the same affection

In most of the cases the lethargy, listlessness and masklike face were not present until the third or fourth week. Several cases, commencing with choreiform twitchings, involving at different times almost all muscles in the body, including the abdominal twitching so characteristic of this group, gradually developed into the lethargic type, with ptosis of one or both lids, diplopia and facial palsies. The patients remained in a state of lethargy and somnolence for several weeks and then, with gradual awakening of the sensorium, the various twitchings reappeared. In two cases the process was repeated three times.

Cranial Nerves.—Involvement of the cranial nerves was not so common in this series as in the earlier epidemic.

First Cranial Nerve: In the first ten cases the olfactory nerve was not examined. After noting small hemorrhages and perivascular infiltration in this nerve in one of the cases examined postmortem, the

Fig. 3 (Case 8).—Spasm of sternomastoid muscle with torticollis.

sense of smell was tested in all patients. It was found markedly diminished in four cases, absent in none.

Second Cranial Nerve: No disturbances of the optic nerve were noted except in two cases. In these the diameter of the papilla seemed enlarged, the veins moderately engorged and tortuous. Around the papilla the retina was slightly opaque and striated. One of these patients died, and microscopic examination showed evidence of optic neuritis. The other patient recovered and six months after the illness all signs of choked disk had disappeared.

Third Cranial Nerve: This nerve was involved in ten cases; diplopia in four; ptosis in six; strabismus in seven. In many the various paralyses were present at different times, but they were not common at the beginning.

Fourth Cranial Nerve: Paralysis was present in three cases.

Fifth Cranial Nerve: Difficulty in mastication was present in two cases, and the masseter muscles did not seem to contract. Neither

patient was lethargic, nor was there pronounced general weakness. In one of these there was hyperalgesia on one side of the face and an abundant crop of herpes along the upper lip. In the other there was diminution of sensibility of the lips and tongue. This patient also had excessive salivation. After six days the anesthesia gradually disappeared.

Sixth Cranial Nerve: Conjugate deviation of the paralytic form was present in four cases.

Involvement of the third, fourth and sixth nerves, as evidenced by total ophthalmoplegia, was present for a short time in two cases.

Seventh Cranial Nerve: Incomplete facial palsy was present in nine cases; in one the paralysis was bilateral.

Eighth Cranial Nerve: Diminution of the acuity of hearing was noted in six cases. Four patients complained of distressing and persistent noises in both ears.

Fig. 4 (Case 11).—Spasm of sternomastoid with torticollis; also paralysis of the muscles of the back of the neck.

Ninth Cranial Nerve: Dysphagia was present in eleven cases, lasting from three days to four weeks. In all cases it was associated with involvement of other cranial nerves, and it was difficult to determine how much the vagus or hypoglossal involvement contributed to the symptom.

Tenth Cranial Nerve: Unilateral flaccid paralysis of the soft palate was noted in two cases. Speech was nasal and deglutition difficult. There was also paralysis of the vocal cord on the same side. Marked gastric irritability and vomiting without apparent cause were present in two cases. Bulimia was present in one case, accompanied by paralysis of the vocal cords and cardiac arrhythmia—extra systole. The bulimia lasted about ten days and gradually disappeared. Irregular respiration was present in one case. It resembled very much the pulse rhythm of a sinus arrhythmia, being mainly trigeminal in character.

Eleventh Cranial Nerve: Persistent spasm of the sternomastoid muscle was seen in one case (Fig. 1). There was paralysis of the trapezius in the same patient and to a still more marked degree in another patient (Fig. 4).

Twelfth Cranial Nerve: Unilateral paralysis of the tongue was noted in one case. Movement of the tongue within the mouth was imperfect on the right side, more so in the attempt to protrude the tongue.

PERIPHERAL NERVES

Pain.—It was the first symptom complained of in sixteen of the cases, most frequently in the face and one side of the neck, next in the extremities. The majority of the patients complained of tenseness and discomfort in the extremities and along the course of various nerves, but at times of sharp and lancinating pains. In one case it was localized in the spine and darted alternately to the right and left. In three patients the lancinating pains were very distressing, frequently sudden and flashing in character and accompanied by mild sensations of contraction. In one patient these pains persisted for four days, requiring morphia to allay them.

Sensory Disturbances.—No dissociation of sensations was observed in eleven of the cases examined for it. Hyperalgesia was noted in three cases. In one it was associated with trophic disturbances, such as blebs and bed sores. No thermo-anesthesia was found.

Trophic and Vasomotor Disturbances.—Persistently cold, clammy and cyanotic finger tips were observed in four patients, profuse sweating in three, excessive salivation in two, decubitus in three.

Tremor.—Coarse tremor was present at some period or other in twenty cases. Its initial location and progress were ascertained in fifteen cases. In six patients it began in one extremity, nearly always a hand, and spread to the lower extremity of the same side; in three it commenced in one side of the face; in two in the abdominal muscles, in one in the muscles of the neck and shoulder, while in the rest it was practically simultaneous in nearly every part of the body.

Slight at first, it became more marked later, so that the affected part would move sufficiently to disarrange the bed clothes. The rate varied. In one patient it was 80 a minute; in others it varied from 20 to 30 a minute. Generally disappearing during sleep, in three cases it persisted, although to a lesser degree. The patient was generally able to diminish or even to suppress it entirely for a few seconds or minutes, when it became entirely uncontrollable. Finer motions and manipulations were not affected very much: and the patients were generally able to feed themselves and write short notes, except during

the height of the disease when the jerkings were extremely violent. Kymographic tracings revealed contractions at irregular intervals but generally of about the same amplitude.

Among the more peculiar and extraordinary spasms were those of the abdominal muscles. The upper part of the recti abdominis and external oblique muscles would contract and twitch, varying from 20 to 50 times a minute. At first the spasm resembled the movements of hiccup but observation showed that it had no relation with respiration or with contractions of the diaphragm. It was generally accompanied

Fig. 5 (Case 15).—Choreiform type of encephalitis. A characteristic position of the hands; it resembles the driving position so frequently found in tetany.

by simultaneous spasm of the extremities and in some instances of nearly the entire voluntary body musculature.

Another extraordinary spasm, in one case, was a spasmodic, spontaneous, dorsal flexion of the right big toe with simultaneous plantar flexion of the small toes. Occasionally fanlike separation of the small toes was seen. I could find no mention in the literature of a similar phenomenon. Tilney and Howe [2] report contractions of the muscles of the big toe, but do not mention any resemblance to the Babinski

2. Tilney, F., and Howe, H. S.: Epidemic Encephalitis, New York, 1920, p. 16.

phenomenon. In my patient the spasm was startlingly regular in frequency, numbering from 53 to 55 a minute. Like the other spasms in the same patient, it persisted during sleep, although to a less degree. This patient recovered. The plantar flexion of the toes ceased first, dorsal flexion of the large toe persisting for some time.

In a few of our patients the position of the hands was extremely interesting. In three they made the "pill rolling" movement so characteristic of paralysis agitans. Two other cases presented the attitude found in tettany (Fig. 5). The arms were held quite close to the chest, the forearms partly flexed, and the wrist flexed, while the fingers were tightly closed over the inverted thumb, simulating the driving position. The position was maintained for many days, and in one patient was present five months after discharge from the hospital. The tonicity of the contracted fingers was evident, but it could be overcome by gentle attempts. One or two of the fingers were generally involved in a continuously intermittent spasm, although the most pronounced spasm was at the wrist. The Trousseau and the Chvostek signs were absent in both cases and neither thyroid, parathyroid therapy, nor administration of calcium affected this tetany-like phenomenon.

Muscles.—Wasting was not a prominent feature but was present to a marked degree in two cases showing evidence of nerve root and cord involvement. The bones of the shoulders, particularly the scapulae, were prominent. There was marked atrophy of the muscles of the back of the neck and shoulder girdle, with occasional wavelike movements or contractions, especially in the trapezius. They showed increased galvanic irritability and anodal closing contraction equal to cathodal closing contraction. Especially in the outer part of the trapezius, the contraction was very slow to the galvanic current. The inner half of the same muscle gave normal galvanic response. There was no pain but a dull aching and sense of fatigue in the involved muscles.

I examined four patients for the myasthenic reaction. In two out of five examinations of the patient in Case 11 great weakness and atrophy of the muscles of the shoulders and back of the neck were found (Figs. 4 and 6).

SIGNS OF MENINGITIS

Sixteen of the twenty-five cases belonged to the "meningoradicular" type of the disease. Rigidity of the neck to a variable extent was present in twelve patients and in two mild opisthotonos. Dilatation of the pupils during the attempt to flex the neck was noticed in two of the patients. The Kernig sign was elicited in six patients, but it was not

constantly present. A typical Babinski sign was noticed in two patients, an atypical Babinski in twelve. The Brudzinski collateral reflex, particularly his "neck sign," was observed in three patients.

MISCELLANEOUS FINDINGS

Enlargement of the spleen was always looked for but never found. Gastro-intestinal disturbance was not common, diarrhea was not observed. Gastric irritability was present in only two patients, both of whom showed evidence of vagus involvement. Obstinate constipation was present in four. Retention of urine was present in two patients—in one of four days' and in the other of six days' duration.

Fig. 6 (Case 11).—Paralysis of the muscles of the back of the neck. Weakness and atrophy of the muscles of the shoulder girdle are also present.

LABORATORY FINDINGS

Blood.—The red blood cells and hemoglobin showed no particular changes. If the disease was protracted, moderate secondary anemia developed. The details of the white blood counts and the differentials are tabulated in Table 1.

Chemical examination of the blood revealed nothing unusual but was an aid in differentiating the condition from other diseases in which nitrogen retention and hyperglycemia occur. As will be seen in Table 2, the blood sugar was normal in spite of the fact that in some of the cases glycosuria was present at the time.

BACTERIOLOGIC FINDINGS

The influenza bacillus of Pfeiffer was isolated from the throat in nine, and from the nose in six, out of twenty patients examined. A

streptococcus was isolated from the throat twelve times and from the nose five times. Blood cultures were made in twenty cases with negative results.

The urine showed a slight trace of albumin in five cases. More than traces were not found, except in two fatal cases.

TABLE 1.—White and Differential Blood Counts in Twenty-Five Cases of Epidemic Encephalitis

Case No.	White Blood Cells	Polymorphonuclears	Lymphocytes	Mononuclears and Transitionals	Eosinophils	Basophils
1	6,400	78	14	7	1	0
2	9,700	73	19	4	4	0
3	6,200	64	25	9	2	0
4	9,100	72	18	8	1	1
5	7,400	69	22	6	3	0
6	6,300	76	14	7	2	1
7	8,100	77	14	7	2	0
8	4,100	60	30	5	5	0
9	10,300	80	15	4	1	0
10	9,600	74	17	7	1	1
11	8,800	68	23	6	3	0
12	10,100	72	23	4	1	2
13	9,000	60	30	5	5	0
14	8,700	84	10	5	1	0
15	6,600	70	18	8	4	0
16	7,400	74	19	5	1	1
17	6,600	76	16	7	1	0
18	8,100	64	26	8	2	0
19	9,100	59	36	4	0	1
20	6,800	77	11	9	2	1
21	7,400	73	13	10	4	0
22	10,200	84	12	3	1	0
23	11,800	80	11	4	5	0
24	13,800	67	23	7	3	0
25	6,500	74	15	8	3	0

TABLE 2.—Blood Chemistry Determinations in Sixteen Cases of Epidemic Encephalitis *

Case No.	Blood Urea	Sugar	Creatinin
1	14	0.12	1.12
2	12	0.07	1.0
3	18	0.09	1.2
4	10	0.11	0.9
5	12	0.12	0.8
6	10	0.07	1.2
7	9	0.09	1.0
8	8	0.08	0.9
9	11	0.12	2.0
10	10	0.13	1.1
11	12	0.07	1.3
12	14	0.09	0.8
13	10	0.07	0.91
14	8	0.08	1.2
15	16	0.06	1.2
16	13	0.10	0.92

* See also Table 4.

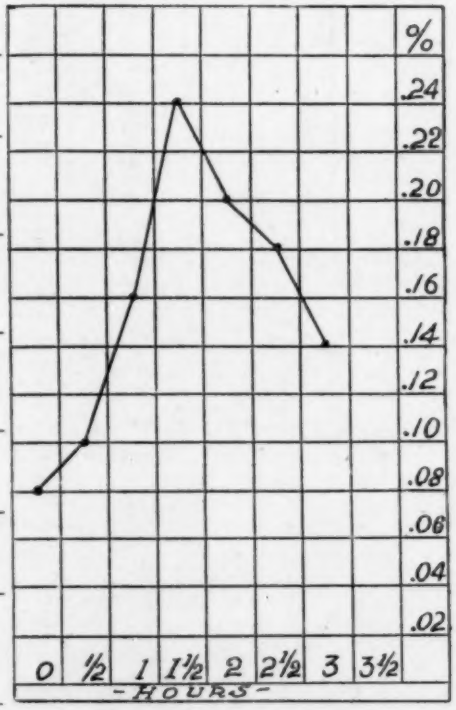
Sugar in the Urine.—A strikingly frequent finding was sugar in the urine. It was found at some period of the illness in nine cases. The amount varied from 0.1 to 1.5 per cent. It was not present through the twenty-four hours, but no definite period, or relation to

time of day or meals could be established. Sugar tolerance tests were made in four cases, and the determination of blood-sugar in sixteen cases.

There was no hyperglycemia in these cases, but all patients except those in Cases 9 and 15 had sugar in the urine at the time of the determinations. Four patients were given 100 gm. of dextrose, and blood sugar determinations were made at one-half hour intervals for three hours. In all, the sugar in the urine was increased, and there was also a considerable increase in the blood sugar which lasted for two and one-half or three hours.

TABLE 3.—SUGAR TOLERANCE TEST IN A CASE OF EPIDEMIC ENCEPHALITIS

Time	Sugar in Urine, Percentage	Sugar in Blood, Percentage	Remarks
8:30	0.1	0.08	Before dextrose
9.00	0.16	0.1	100 gm. dextrose given at 8:30 a. m.
9:30	0.5	0.16	
10:00	1.0	0 24	
10.30	1.0	0.2	
11:00	0.5	0.18	
11:30	1.16	0.14	



Other Elements in the Urine.—Nothing else abnormal, of any significance, was found. Acetone was present in two patients in both of whom it was associated with marked vomiting. Hyaline casts were present in the urine of the two patients whose urine had a moderate cloud of albumin.

SPINAL FLUID

The spinal fluid was clear in all cases. It was under moderately increased pressure in seventeen of the twenty-one patients on whom

Fig. 7 (Case 20).—Base of the brain showing marked hyperemia and a moderate exudate, particularly in the region of the pons and medulla.

lumbar puncture was performed. A trace of globulin was present in all cases, and in ten there was a marked precipitate. The cell count was below 10 in two cases. No organisms were found, and the Wassermann reaction was negative in all. Further details are given in Table 4.

MISCELLANEOUS LABORATORY FINDINGS

Chemical.—Microscopic and bacteriologic examination of the stools in twelve of the cases showed nothing unusual, and the same applies to gastric analysis made in four cases. The Widal test, performed in sixteen cases, showed no agglutination.

GROSS AND MICROSCCPIC PATHOLOGY

The pathologic findings were practically confined to the central nervous system. In one case definite granular degeneration and cloudy

TABLE 4.—THE FINDINGS IN THE CEREBROSPINAL FLUID IN TWENTY-ONE CASES OF EPIDEMIC ENCEPHALITIS

Case No.	Color	Fehling's Reduction	Globulin	Cell Count	Polymorphonuclears	Lymphocytes	Bacteria	Wassermann Reaction
1	Clear	Normal	++	27	15	85	0	0
2	Clear	Normal	+++	21	7	93	0	0
3	Clear	Normal	++	30	10	90	0	0
4	Clear	Normal	+	22	13	87	0	0
5	Clear	Normal	+	14	14	86	0	0
6	Clear	Normal	++	17	10	90	0	0
7	Clear	Normal	+	9	13	87	0	0
8	Clear	Diminished	+	21	12	88	0	0
9	Clear	Normal	++	25	10	90	0	0
10	Clear	Normal	++	32	9	91	0	0
11	Clear	Normal	++	41	13	87	0	0
12	Clear	Normal	+	12	7	93	0	0
13	Clear	Diminished	++	26	2	98	0	0
14	Clear	Normal	++	17	5	95	0	0
15	Clear	Normal	+	9	10	90	0	0
16	Clear	Normal	+	10	7	93	0	0
17	Clear	Normal	+	14	13	87	0	0
18	Clear	Normal	+	16	14	86	0	0
19	Clear	Normal	++	17	6	94	0	0
20	Clear	Normal	+++	57	11	89	0	0
21	Clear	Diminished	+	22	7	93	0	0

swelling were present in various organs, but the patient had a persistently high temperature which lasted for five days. The results of postmortem bacteriologic examination of the heart blood, various viscera, brain and meninges were negative.

We had occasion to examine two fetuses—both from spontaneous abortion. One was of eight months' gestation, the other of four and one half. In the older the meninges were normal, but the pia-arachnoid showed edema and congestion. There were microscopic hemorrhages in various parts of the brain and spinal cord, particularly at the base of the brain, and in the pons. Capillaries and veins were engorged and dilated, but there was no perivascular infiltration. There was also

cellular degeneration, but no inflammatory changes. The whole was what one would expect in a fetus that has been dead in utero for some time. Findings in the younger fetus were similar.

In the adults the findings were the same as those described by other observers and by Allen and myself.[1] These cases, it will be recalled, were characterized clinically by marked restlessness, choreiform jerking and peculiar twitchings of the abdominal muscles, and

Fig. 8 (Case 20).—Base of the brain; round cell infiltration of the superficial layer; perivascular infiltration of the blood vessels.

were designated as the "meningoradicular" type. In addition to the usual findings in the brain substance, they showed pronounced changes in the meninges and spinal cord indicating that the disease was a meningo-encephalo-myelitis.

In two of our cases the meninges showed a marked degree of inflammation. The blood vessels were engorged and dilated, and there was a considerable amount of exudate at the base of the brain (Fig. 7).

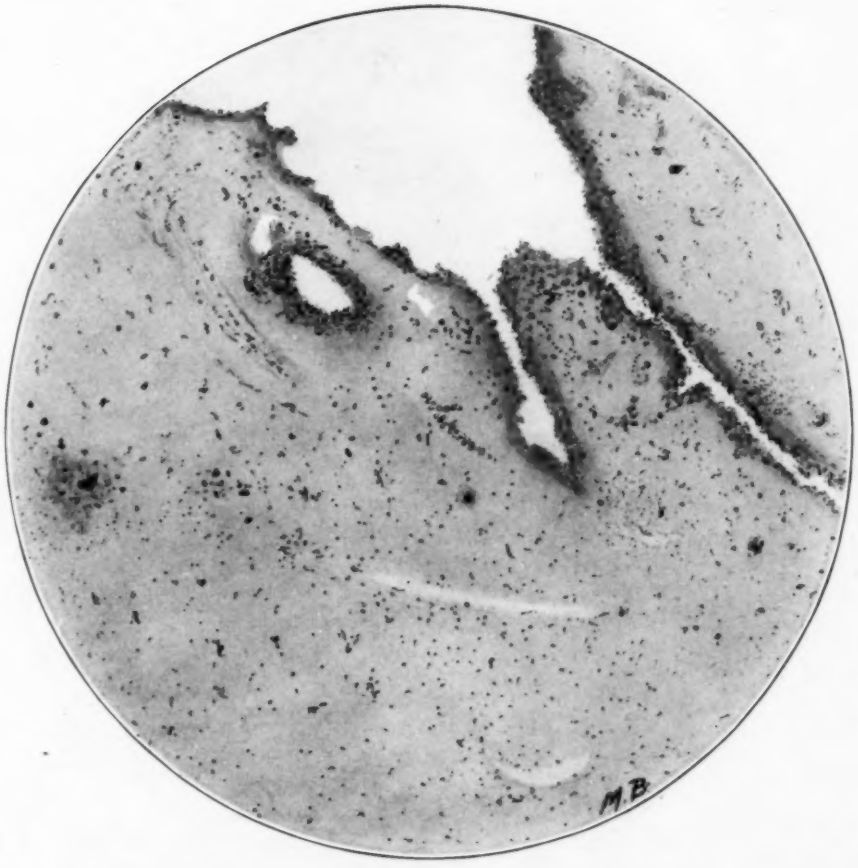


Fig. 9 (Case 24).—Base of the brain showing inflammatory involvement of the brain tissue just underneath the pia arachnoid. Note the diffuse cellular infiltration and the perivascular infiltration of the blood vessels.

The pia was thickened and grayish-red. Smears and cultures from the exudate were invariably negative. On section tiny red spots were seen scattered throughout the brain substance but were most noticeable at the base, in the pons and the cerebral peduncles, where some of the spots were quite large, being from 2 to 3 mm. in diameter. Minute hemorrhages were scattered throughout the cerebral cortex, basal ganglions, brain stem and spinal cord. The ventricles were slightly dilated and the ependyma thickened.

Microscopically the meninges showed marked inflammation, collections of inflammatory cells being found in divers parts. They were particularly frequent about the smaller blood vessels. Under high power these cells were found to be lymphocytes and mononuclears. Occasionally a polymorphonuclear leukocyte was seen in the adventitial lymph spaces.

The cortex showed slight dilatation of the capillaries and moderate cloudy swelling in some of the motor cells. The cells were swollen, the nuclei eccentric and there was some satellitosis.

At the base there was an unusual degree of inflammatory reaction just beneath the pia-arachnoid. In this region many accumulations of lymphoid cells were seen (Figs. 8 and 9). There was also diffuse infiltration with inflammatory cells. The vessels were dilated and showed marked perivascular infiltration with lymphocytes and mononuclears. The nerve cells in some places stained poorly, with some chromatolysis. In one case there was distinct inflammatory involvement of the olfactory bulb and optic nerve. The axis cylinders were swollen and stained freely and in some places were disintegrated. The protoplasm around the nuclei was increased in amount, and the nuclei were acentric. The blood vessels were congested and surrounded by mononuclear cells. In some places collections of lymphocytes and mononuclear cells were found at some distance from the blood vessels.

CLASSIFICATION

The twenty-five cases may be divided into five clinical types: (1) the lethargic type (one case), (2) the meningoradicular type (thirteen cases), (3) the paralysis agitans type (six cases), (4) the poliencephalitis type (two cases), and (5) the choreiform type (three cases).

The lethargic type was characterized by profound somnolence, cranial nerve palsies, especially of the third nerve, diplopia and masklike facies.

The meningoradicular type was marked by headache, cloudiness of sensorium, stiffness of the neck, pain along the course of the large nerves and by restlessness and twitching, often peculiar twitching of the abdominal muscles. A suggestive Kernig's sign and a pseudo or

true Babinski sign were frequently seen. The spinal fluid generally had more pronounced increase of globulin and cells. Necropsy examination generally revealed more inflammation of the meninges and contiguous brain substance than in the other types.

The paralysis agitans type was characterized by a typical parkinsonian attitude and expression, various tremors and the characteristic position of the hand with "pill rolling" motion. The voice was monotonous and generally feeble. The gait was characteristic, and the patients sometimes volunteered the statement that they could run better than they could walk.

The poliencephalitis type was characterized by paralysis of one or more cranial nerves, most frequently the third and seventh. Lethargy and stupor were not prominent but marked asthenia was a feature. These cases suggested myasthenia gravis, and one patient gave a positive myasthenic reaction.

In the choreiform type the most pronounced disturbances were the extreme and persistent spasms. The patient blinked his eyes, jerked his head, twitched the mouth and made grotesque grimaces and contortions. One side of the body was generally more involved than the other. Cranial palsies were not prominent and were transient. The fever was generally quite high. Speech was thick and indistinct and deglutition difficult. The movements persisted even during sleep, although to a much less extent. Mild delirium was generally present at night but subsided toward morning.

PROGNOSIS AND CONVALESCENCE

Of the twenty-five cases in this series, four were fatal. Death apparently was due to involvement of the medullary centers. It was invariably preceded by marked disturbances in deglutition and respiration. Aggravation of the patient's condition frequently was rapid. Exceedingly few complications were seen.

Convalescence was generally slow. Exacerbations and remissions were frequent. The average duration of the disease in the mild cases was four weeks, of the moderately severe cases, about two months and of the severe cases, from three to five months. While progress is slow, it is constant. Two cases early in the epidemic were diagnosed as multiple sclerosis.

Of the twenty-one patients that survived, twelve are completely well. Five complain of lack of strength and endurance, nervousness, slight tremor and unsteadiness when excited or after moderate exertion, but they are doing some light work. The remainder are still more or less invalided—one fourteen months, and the other three, from nine to ten months after the onset of the illness. Their complaints are the same as those mentioned except to a more marked degree. Two of

the patients also have cold and clammy hands and slight cyanosis of the finger tips. Three patients complain of excessive sweating and also have grooving of the nails. In two cases shedding of the nails took place.

The patients of the paralysis agitans type showed the slowest improvement. Nevertheless, of the six patients belonging to this group, all but two are well at the present time (nine to ten months after illness). Of the two, one is able to walk about and is doing a moderate amount of work. The second is still confined to bed during the greater part of the day, and after the slightest exertion exhibits generalized tremor and the characteristic position and tremor of the hands.

TREATMENT

Specific Treatment.—During the latter part of the epidemic I used convalescent serum in two cases. Twenty-five c.c. were injected intravenously, repeated in twenty-four hours. Both patients showed improvement soon after. The temperature came down to normal during the following three days and remained normal. The convalescence, however, did not seem to be shortened and the tremor, nervousness and twitchings persisted for six weeks in one case and for nine weeks in the other. The white blood count and the differential count showed no appreciable change following the injection of the serum.

General Treatment.—General care was relied on in the majority of cases. The most troublesome symptom in many cases was the dysphagia. It rendered feeding of the patient exceedingly difficult and also endangered life by the possibility of aspiration pneumonia or by suffocation. In three cases esophageal feedings were resorted to, in one case for three weeks. The patient made a complete recovery.

Sedatives.—Hypodermic injections of magnesium sulphate were tried for the twitchings and spasms but were of no benefit. Benzylbenzoate was also ineffective. Bromids and chloral were effective in large doses. Hypodermic injections of morphin and hyoscin were the most satisfactory and dram doses of paraldehyd were next in efficiency.

After the acute stage electricity and massage were of definite value.

SUMMARY

The symptoms of the patients seen in the latter part of the epidemic varied greatly from those seen early. These symptoms were restlessness, twitching and insomnia, but the transition stages and the passing of the patients in a number of these restless and twitching cases into a state of lethargy with palsies of the cranial nerves show that they are cases of lethargic encephalitis. Laboratory and necropsy findings point to the same conclusion.

While there was no relation between influenza and epidemic encephalitis in the individual cases, there was a suggestive relation between them in the community.

The symptomatology was extremely varied and resembled that of a great many neurologic diseases.

The pathologic findings consisted of minute hemorrhages in various parts of the nervous system with perivascular infiltration of the capillaries and small blood vessels with round cells and lymphocytes. In the cases belonging to the meningoradicular type these changes were apt to be prominent and considerable involvement of the meninges and superficial brain tissues was frequently present. The blood frequently showed slight eosinophilia. Sugar was frequently present in the urine. There was no hyperglycemia, but the sugar tolerance was lowered.

The mortality in this series was 16.80 per cent. The patients that survived generally had a protracted convalescence, but all showed a definite and progressive improvement.

HISTORY OF LUMBAR PUNCTURE (RACHICENTESIS)

THE OPERATION AND THE IDEA

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The credit for lumbar puncture has been given in most historical reviews to one man, not always the same, generally either Corning or Quincke or Winter. The decision, however, does not seem really so simple. Many men have contributed important ideas in the growth of this subject as we know it today, and the credit given seems to have depended largely on the particular interest of each writer. I have, therefore, made an attempt to show the source of each successive new idea in this field, without in general including confirmatory work, important though that may have been.

Swifft, 1774, was stated by Wyss, 1893, to have punctured the cerebral ventricles, and the same writer noted that Wernicke and Zeuner, 1841, used the same procedure after trepanning. I have not been able to locate the original works in either the first or second series of the Index Catalogue of the Library of the Surgeon General's Office. Middeldorpf, 1856, performed a cerebral puncture on the living through a trephining, according to Levinson, 1918. Quincke, 1872, in studying the physiology of the cerebrospinal fluid in dogs, put a cannula into the subarachnoid space of the spinal cord in the upper lumbar region, and injected 1 c.c. of mercury sulphid using painters' finest granulated cinnabar rubbed up into an emulsion with Zuckerwasser and diluted with 5 to 10 volumes of water. He did not examine the fluid, however, but only the distribution of the red granules postmortem. Naunyn and Schreiber, 1881, trepanned the parietal bone and inserted a cannula into the brain, and also into the subarachnoid sac of the cauda equina, but did not make a direct puncture.

Corning, 1885, with a view to determining whether local anesthetization of the spinal cord was within the range of practical achievement, laminectomized a frog, then put a hypodermic needle through the membranes of the medulla spinalis. He concluded that this procedure was not practicable without removal of the arches of the vertebrae on account of the danger of wounding the cord, and therefore decided to inject the anesthetic (cocain hydrochlorate) between the spinous processes of the lower dorsal vertebrae. His two protocols, one on a dog and one on a man, show that anesthesia of the hind legs was in fact produced. Besides this, apparently the first direct spinal puncture, his title "Spinal

Anesthesia and Local Medication of the Cord" indicates that he was the first to perform intraspinal injection.

Rehn, 1886, demonstrated a child whom he had cured of hydrocephalus by "puncture," but he did not describe the nature of the puncture.

Wynter, 1891, somewhat as Naunyn and Schreiber did ten years before, punctured the lumbar spine and inserted a cannula on the brain. The object was continuous drainage to relieve hydrocephalus. Wynter also quoted both Hulke and P. Gould as having carried out the same procedure, after laminectomy. The author cited, Essex Wynter, appears to be that "Essex and Winter," to whom Déjerine, 1914, erroneously gave the credit for introducing puncture clinically.

USE OF LUMBAR PUNCTURE IN DIAGNOSIS

Quincke later, in 1891, first worked out a serviceable technic for clinical lumbar puncture, and since then, in the words of Nonne, 1915, "a new science has arisen." Quincke was, furthermore, the first to notice "lymph-corpuscles" in the spinal fluid. He was also the first to mention the possibility of using lumbar puncture in diagnosis, but only from the point of view of pressure and volume relations. These have remained relatively fruitless for diagnosis, though recent work is more encouraging, such as that by Sharpe, 1920, Sidbury, 1920, and Ayer (unpublished).

Lichtheim, 1893, first noted the grossly purulent and the bacteriologic nature of the fluid in meningitis following otitis media due to streptococci and pneumococci.

Von Ziemssen later, in 1893, similarly noted that "the diagnostic value of lumbar puncture is great, especially in differentiating the kinds of meningitis," and added: ". . . Just as in puncturing pleurisies." In this last analogy he antedated Widal and Ravaut, 1900, but they were instrumental in actually developing the idea by the discussion of the various kinds of cells. Von Ziemssen also seems to have been the first to report that lumbar puncture relieved headache, based on his observations on tuberculous, cerebrospinal and purulent meningitis. Von Ziemssen, like Corning, contemplated intraspinal treatment, when he said in relation to epidemic cerebrospinal meningitis: "It even seems not impossible to introduce directly curative substances in solution into the vertebral canal and so inaugurate a local therapy of the spinal cord and brain." Still a third, independent, and for a time neglected, prophet of lumbar puncture "as a means of applying medication directly to the spinal meninges," was Browning, 1894. This idea has since been put in practice by the brilliant work of many investigators.

Freyhan, 1894, seems to have been the first to describe the microscopic sediment of the cerebrospinal fluid as containing pus corpuscles and tubercle bacilli in a case which has become classic, because it again yielded tubercle bacilli in a second lumbar punctate a week later; the patient, however, made a complete recovery. A cynic has suggested that this observer misinterpreted scratches on the glass slide.

Fürbringer, 1895, in a case of tuberculosis drew off at one puncture 110 c.c., which seems a record, though the author made no such claim. Surgeons since that time have reported a liter a day draining from skull fractures.

Heubner, 1895, was the first to dare to emphasize the inestimable value of lumbar puncture "for the practitioner."

Goldscheider, 1895, independently of Corning, 1885, but in pursuance of von Ziemssen's vision of therapy, injected cocain intraspinally to relieve painful contractions due to compression myelitis, but without success.

Wentworth, 1896, in this country, found many lymphocytes in the cerebrospinal fluid of tuberculous patients, an observation often but erroneously quoted since (by Duflos, 1901; Variot, 1903; Kopetzky, 1906) as the beginning of cytology in relation to meningeal affections. The work, however, proved an important stimulus to others.

Rieken, 1895, in Quincke's clinic at Kiel was the first to perform spinal puncture in a syphilitic patient. He noted albumin but no cells, although at necropsy there was extensive softening of both hemispheres and localized pachymeningitis.

Korczyriski and Wernicki, 1896, wrote a paper on the importance of lymphocytes in serous effusions of the pleura, and related them to tuberculosis; while Bernheim and Moser, 1897, described the preponderance of mononuclears over polymorphonuclears in tuberculous meningitis, as contrasted with the reverse proportions in epidemic and purulent meningitis. These authors seem to antedate the claims of Widal and his associates, although Widal, 1901, denied the priority and the significance of the work of Korczyriski and Wernicki.

Bier and Hildebrandt, 1899, with the object of anesthetizing a larger area than could be reached by the intradermic infiltration anesthesia of Orloff, 1887, Wölfler, 1887, Link, 1888, Reclus and Wall, 1889, and Schleich, 1892 and 1894, or by the regional anesthesia of Fraenkel, 1887, Pernice and Oberst, 1890, cocainized the spinal cord through a lumbar puncture needle. This work was done without knowledge of the work of Corning, 1885, and of Goldscheider, 1895. Later Bier, 1901, though mentioning Corning denies him priority! None the less, Bier's contribution seems to be the first practical application of the idea to surgery.

Lewkowicz, 1900, showed that lymphocytosis was present in 80 per cent. of tuberculous meningitis, and seems to have been the first to suggest that syphilis (tabes or general paralysis) might also show lymphocytosis, though he did not give any evidence.

Sicard, 1900, devoted a whole thesis to intraspinal injections, discussing prior anatomic and physiologic studies on the cerebrospinal fluid, together with his own therapeutic injections of morphin, serum and other substances. He seems also to have suggested the use of intraspinal mercury, not for immediate spirocheticidal effect but in order to increase the permeability of the meninges to such drugs as were caused to circulate in the blood.

Marinesco, 1900, suggested intraspinal treatment of meningomyelitis with methylene-blue in artificial serum.

LUMBAR PUNCTURE IN SYPHILIS

Widal, Sicard and Ravaut, 1901, reported their observations, now classical, on lymphocytosis in the cerebrospinal fluid of tabetic and general paretic patients, with negative findings in paralysis agitans, chorea, hemiplegia, and other nerve conditions as controls. They drew the conclusion that lymphocytosis is "no more specific to meningoencephalitis and tabes than to tuberculous meningitis." This view may be said to have been generally accepted, with perhaps the qualification contained in the statement of Babinski and Nageotte. Nageotte, 1901, made the important observation "there may exist in certain patients . . . a very slight syphilitic meningitis, inappreciable clinically, but well able to give lymphocytes in the cerebrospinal fluid . . . I have seen such a case." The idea was developed by Joffroy, 1901, Widal, 1902, and Ravaut, 1903. Nageotte failed at this time, however, to draw the inference that lumbar puncture should often be done despite the absence of appreciable clinical signs of meningitis. It remained for Babinski and Nageotte in 1901 to make the first statements:

1. Though there be an augmentation of lymphocytes in tuberculous meningitis, the diagnostic value of this sign remains complete from the viewpoint of diffuse syphilis, for it is hardly probable that doubt in diagnosis should ever arise between tuberculosis and tabes or paresis.

2. It would be interesting to puncture syphilitics systematically.

3. Besides lymphocytosis in the cerebrospinal fluid the only symptom may be an Argyll Robertson pupil.

Sicard was credited by Widal, 1901, with having stated the importance of using a fine and sharp needle in order not to tear the dura and thus allow leakage and consequent headache. Sicard reported this himself in 1902, and more recently the same subject has been discussed by McRoberts, 1918, and Wegeforth and Latham, 1919.

Milian, Crouzon, and Paris, 1902, indicated the importance of regarding headache in syphilitic patients as an indication for lumbar puncture, even in the absence of other symptoms attributable to the nervous system, because in two such cases they found an excess of lymphocytes.

Widal and Le Sourd, 1902, first reported a cure of a syphilitic lymphocytosis by the usual general antisyphilitic treatment.

Ravaut, 1903, emphasized the latent period silent clinically as the reason for his belief (which resembles that of Babinski and Nageotte, 1901) that "perhaps even before any nervous manifestation systematic cytologic examination should be made," and as evidence he stated that in a hundred and eighteen patients in the secondary stage, only seven of whom showed nerve signs, such as iritis or facial paralysis, there were excessive cells in the cerebrospinal fluid in all.

Schittenhelm, 1903, introduced the useful though not always strictly accurate terms for incipient cerebrospinal syphilis "monosymptomatic and oligosymptomatic pictures of disease or forme frustes"; and Ravaut, 1904, described lumbar puncture aptly as "veritable biopsy of the central nervous system."

Wassermann and Plaut, 1906, seem to have been the first to examine the spinal fluid of syphilitic patients by the Wassermann reaction. Similar work was carried out by Levaditi, Ravaut and Yamanouchi, 1908, who concluded that the complement-fixation method may up to a certain point be of service in the early diagnosis of nervous localizations of syphilis, and that pleocytosis is often present without the presence of lipoids which produce the Wassermann phenomenon.

Kopke, 1906 and 1907, seems to have been the first to inject arsenic intraspinally as a spirocheticide, treating sleeping sickness with atoxyl, on the basis of Castellani's discovery of *Trypanosoma gambiense* in the cerebrospinal fluid.

Ravaut, 1907, expressed the important conception that in the secondary stage syphilis behaves like a real septicemia.

Kausch, 1908, reviewed the needles, manometers and technic in general of Quincke, Von Arlt, Bier, Grosse, B. Krönig, G. Krönig, Pochhammer, Tomaschewski, Wilms, and Nemenow, and proposed a new instrumentarium.

Quincke, 1909, said that in syphilis cells occur in the fluid earlier and more frequently than in other "serous meningitides."

Alt, 1910, reported treatment of neurosyphilis by the injection of arsphenamin intramuscularly, and Iversen, 1910, intravenously, but neither examined the cerebrospinal fluid. This was then done by Sicard and Bloch, 1910, who found the Marsh ring test negative after

intramuscular injection of arsphenamin, but positive after intravenous injection, wherefore they recommended the latter route.

Taege, 1910, first reported treatment of a baby by milk from the mother who had been treated with arsphenamin, and similarly Meirowsky and Hartmann, 1910, cleared up skin lesions in a child by subcutaneous injection of serum from patients treated with arsphenamin. Marinesco, 1911, was the first to report such arsphenamized serum (in vivo) intraspinally, but the first thorough appreciation of the ideal value of this preparation and the first real success with it was that reported by Swift and Ellis, 1912 (and 1913), then by Robertson, 1913.

Swift and Ellis, 1912, also tried direct intraspinal injection of arsphenamin in monkeys and thought it too dangerous to use, but injected 0.5 mg. and 1 mg. of neo-arsphenamin directly without ill effect. Wechselmann, somewhat later in 1912, used both arsphenamin and neo-arsphenamin intraspinally; Ravaut, 1913, used neo-arsphenamin intraspinally; Marinesco and Minea, 1914, also used neo-arsphenamized serum in vitro intraspinally, as did Ogilvie, 1914, and Swift, 1916.

Ravaut, 1911, emphasized that the "Wassermann reaction in the blood is often negative when gross nervous lesions are in process of evolution," and insisted on lumbar puncture without waiting for clinical symptoms.

Fraenkel, 1912, by use of the Auswertungsmethode of Hauptmann, 1910, that is, by increasing the amount of fluid from the original 0.2 c.c. to 1.0 or 1.5 c.c., found the Wassermann reaction positive in asymptomatic neurosyphilitics whose condition was similar to that of patients in whom other observers had reported negative reactions with the use of only 0.2 c.c.

Ravaut, 1913, first attempted intraspinal treatment with mercuric chlorid and cyanid. Pussen, 1913, and Marie and Levaditi, 1913, suggested the use of mercuric chlorid intracranially. Byrnes, 1914, used serum mercurialized in vitro.

Pollitzer, 1913, thought it likely that 100 per cent. of cases of syphilis would show central nervous system involvement at some early stage . . . and that discovery of this involvement depended on the methods employed. In the ensuing discussion Wile agreed.

Wile and Stokes, 1914, recommended routine lumbar puncture on syphilitic patients in primary or secondary stages. In 1915, they reported six cases of primary syphilis with abnormal cerebrospinal fluid in four.

Wile and Stokes, 1915, reported the case of a patient (Case 5) with a chancre only about one week old, yet with a positive spinal fluid.

Similarly, Read, 1915, found, only two weeks after the appearance of a chancre, which appeared twenty-three days after exposure, that the cerebrospinal fluid was markedly pathologic.

Solomon, 1916, defined "latent syphilis" as:

> A condition in which there are no discernible lesions or manifest symptoms except the positive Wassermann reaction. Syphilis may be considered a disease of remissions and exacerbations, the latent period merely a remission, following which a severe lesion is to be expected. Therefore when we treat a patient having a positive Wassermann reaction during the period of latency, we are not treating the reaction but rather the patient for the purpose of curing him of a chronic disease and thus preventing a flare-up.

He therefore affirmed that in every case showing a positive Wassermann reaction a lumbar puncture is indicated for the purpose of examining the spinal fluid. This appears to have been the earliest absolute requirement of puncture for any syphilitic condition.

Southard and Solomon, 1916, introduced the term paresis sine paresi for neurosyphilis without clinical symptoms or signs but with laboratory signs.

Thompson also, in 1916 and 1920, "urged all syphilitics, no matter at what time in the course of the disease they are seen, to submit to a spinal puncture."

Lind, in Hazen's excellent book on "Syphilis," 1919, stated that "lumbar puncture should be insisted on to the patient and his relatives whenever there is the slightest suspicion of central nervous system involvement."

Wile, 1917, reported:

> At the University Hospital (University of Michigan) specimens of spinal fluid are withdrawn from every patient having syphilis on entrance into the hospital, as a routine diagnostic measure. Many unsuspected cerebrospinal involvements have been discovered by this routine procedure.

Keyser, 1917, writing on "Syphilis of the Nervous System Without Characteristic Symptoms and Signs" made the epigram:

> The absence of characteristic symptoms or signs does not exclude syphilis . . . . When in doubt suspect syphilis, and when not in doubt still suspect syphilis . . . All patients in whom syphilis of the central nervous system is suspected should be subjected to lumbar puncture.

Rosen, 1917, stated that the medical profession in general should recognize the necessity of early lumbar puncture as a routine measure in all cases of syphilis, and continued:

> At the Vanderbilt clinic it is our aim to puncture all . . . cases early, that is, after about the third month of their infection . . . Up to a year ago we were content to defer lumbar puncture, unless clinical signs of neurosyphilis were present, until just before the dismissal of the patient as cured.

Corbus, 1917, recommended that "lumbar puncture with spinal fluid examinations, be demanded in all cases of syphilis, no matter what the stage . . ."

Dennie and Smith, 1918, concluded that the cerebrospinal fluid of patients with early treated syphilis should be examined before the treatment is finished, as 50 per cent. of their patients had positive findings in the cerebrospinal fluid without signs or symptoms of involvement of the central nervous system.

Sterne, 1918, insisted that the nervous system is invaded early, and not late, as is generally supposed; and that examination of the cerebrospinal fluid along with the examination of the blood, in the early stages of the disease, is far more important than in the later stages, although such an examination is not ordinarily made.

Kaliski and Strauss, 1918, stated that: "Cerebrospinal syphilis occurs comparatively soon after infection. In patients in whom the Wassermann reaction in the blood persists, and in latent or tertiary cases in which treatment has been neglected . . . lumbar puncture should be done."

Wohlwill, 1918: "Today it must be regarded as the exception when in the stage of the general septicemia the central nervous system is not involved"; though he did not define the period of septicemia.

Frothingham, 1919: "It seems fair to demand that in all cases of old syphilis a lumbar puncture should be performed as a diagnostic procedure before instituting treatment"; and, in discussion of Frothingham's article before the Association of American Physicians, Collins made the rather reactionary reservation that "lumbar puncture will not reveal syphilis of the central nervous system as well as careful examination."

Fildes, Parnell and Maitland, 1919, recommended that the cerebrospinal fluid be examined in every case of syphilis.

Neu, 1919, recommended:

> Utilizing the serologic findings of the spinal fluid as well as of the blood in every case of syphilitic infection when first contracted and not waiting until there is other evidence of the nervous system being affected.

Eskuchen's book, Die Lumbalpunktion, 1919, gives a large amount of material, but not especially conveniently digested.

Larkin and Cornwall, 1919, demanded that study of the spinal fluid be made in every case of syphilis, as 30 per cent. of the patients show some pathologic change.

Fordyce, 1919, with his colossal experience, affirmed that more patients have serologic syphilis without objective signs than signs of syphilis without serology, hence serology is more reliable than signs.

Scott and Pearson, 1920, believe that every patient who shows a positive Wassermann reaction in the blood and every patient with late or asymptomatic syphilis should receive at least one examination of the central nervous system during the early part of his treatment. In at least eight of their 210 cases the central nervous system was involved in considerably less than a year. In fact, they feel so strongly as to say that "To be satisfied with a neurologic examination of a syphilitic as proof whether cerebrospinal syphilis exists or not, is almost criminal."

Stokes and Brehmer, 1920, remarked on the "weakness of unaided clinical judgment in the recognition of the disease," and proceeded to demonstrate in a group of thirty-nine syphilitics intensively studied that 80 per cent. had involvement of the central nervous system.

Guy, 1920: "Before any patient with syphilis is finally discharged a lumbar puncture is made."

Fordyce, 1921: "The activity of a syphilitic process in the central nervous system is indicated much earlier, more accurately and often only by the cytologic changes in the spinal fluid which should always supplement the physical examination."

SUMMARY

Generally, résumés of this field give the impression that Quincke is responsible for the technic, and that for the interpretation of cerebrospinal fluid pathology the credit is preponderantly due to Widal and Ravaut. I have attempted to trace systematically all the investigators who have initiated the successive ideas in the development of lumbar puncture,[1] and particularly to indicate the increasing insistance on early lumbar puncture.

1. The details of references have been omitted, since they may easily be found in the Index Medicus, the Index Catalogue, or in one or other of the authoritative textbooks, such as Hazen's.

THE PREPARATION OF COLLOIDAL GOLD SOLUTION FOR TESTING SPINAL FLUID*

ALEXANDER O. GETTLER, PH.D., AND J. W. JACKSON, M.D.
NEW YORK

Lange's colloidal gold reaction has been found to be a highly satisfactory aid in the diagnosis of such diseases as paresis, tabes, syphilitic leptomeningitis and the like. At Bellevue Hospital, for some years past, we have been using this test as a routine measure in conjunction with the Wassermann reaction, and the result of a comparative study of the two will be published at another time. Suffice it to say for the present that, in our experience, the colloidal gold reaction is as reliable in the diagnosis of cerebrospinal syphilis as is the Wassermann, if not more so. That the test is not more widely employed is due, we believe, to technical difficulties, since even though great pains are taken, a considerable percentage of all solutions prepared are found to be useless, either because of cloudiness or excessive variations in sensitiveness. After experimenting extensively with various methods, we have found the following procedure to yield the most satisfactory results:

1. We employ 1.5 liter Florence flasks for the preparation of liter quantities. Ordinary laboratory glassware suffices; special glass is not essential. As far as cleaning the glassware is concerned, any procedure which results in a clean utensil is permissible. We prefer to treat the flasks with aqua regia and then wash them carefully several times with distilled water.

2. Triply distilled water is not necessary, provided the following procedure is followed: To 10 liters of water in a large copper still, add a few crystals of potassium permanganate and distill, rejecting the first 300 c.c.

3. In the matter of reagents, the ordinary chemically pure substances give satisfactory results.

4. The temperature may be raised either slowly with a Bunsen burner or rapidly with a rose burner, and it matters little in what sequence or at what temperature the reagents are added.

5. In our experience, contrary to that of others, we have not found it necessary to titrate the strength of the formaldehyde nor to measure carefully the amount added, but are convinced that the amount of

* From the Chemical Laboratory, Department of Pathology, Bellevue and Allied Hospitals and of the University and Bellevue Hospital Medical College, New York.

formaldehyde used and its reaction make little difference. We use ordinary concentrated formaldehyde as purchased (40 per cent.) and add about 0.2 to 0.3 c.c. to each liter of solution.

6. An important factor, however, is the care with which the mixing is done. Rapid and thorough mixing are necessary. We advise shaking before adding the formaldehyde, and to add it while the solution is still in motion; then shake thoroughly for from one-half to one minute.

THE METHOD IN DETAIL

Into a clean 1.5 liter Florence flask place 1 liter of water, distilled as outlined in the foregoing. To this add 10 c.c. of 1 per cent. gold chlorid solution, 7 c.c. of 2 per cent. potassium carbonate and 0.5 c.c. of 1 per cent. oxalic acid. Heat this mixture to the boiling point, and at this temperature remove the flask from the flame, holding it by means of a towel and shake vigorously. While the solution is still in motion, add quickly from 0.2 to 0.3 c.c. of ordinary concentrated 40 per cent. chemically pure formaldehyde and at once shake thoroughly for from one-half to one minute. After from three to four minutes the color usually commences to develop. If, however, there should be no indication of color, the solution must again be shaken well and, while still in motion, an additional 0.1 to 0.2 c.c. of formaldehyde quickly added; this addition almost invariably produces the desired change. At no time during this process should the shaking be stopped. The color should develop rapidly to a deep red. If, however, there is a delay in the appearance of color, the mixture should be allowed to stand, and in a minute or two the color will start to develop; at this instant the solution should again be thoroughly shaken until the color reaches a deep red shade. The entire process, from the moment the solution reaches the boiling point until the color is fully developed, requires at the most about three minutes.

News and Comment

ANNUAL MEETING OF THE AMERICAN PSYCHIATRIC ASSOCIATION

At the seventy-seventh annual meeting of the American Medico-Psychological Association, held in Boston, May 31 to June 3, a new constitution was adopted, which provided—among other slight changes—a change in the name of the organization, which will hereafter be known as the American Psychiatric Association. *The American Journal of Insanity* will hereafter be the official organ of the association, and will be published under a new name—*The American Journal of Psychiatry*. The former transactions bound in book form will be omitted, but all members and Fellows will be furnished with the journal.

The Association by precedent nominates its retiring vice president for the presidency, but Dr. Sanger Brown requested that his name be withheld from the nomination, and the following officers were elected: Dr. Albert M. Barrett, Ann Arbor, Mich., president; Dr. H. W. Mitchell, Warren, Pa., vice president, and Dr. C. Floyd Haviland, Middletown, Conn., secretary-treasurer.

The time and place of next year's meeting have not been decided.

Abstracts from Current Literature

EIN NEUER CEREBRALER SYMPTOMKOMPLEX (ISOLIERTER AUSFALL DER MIMIK, PHONATION, ARTIKULATION, MASTIKATION UND DEGLUTITION BEI ERHALTENER WILLKUELICHER INNERVATION DES ORALEN MUSKELKOMPLEXES) (A NEW CEREBRAL SYNDROME [ISOLATED LOSS OF MIMIC, PHONATION, ARTICULATION, MASTICATION AND DEGLUTITION WITH RETENTION OF THE VOLUNTARY INNERVATION OF THE ORAL MUSCLE-COMPLEXES]). OSKAR FISCHER, Med. Klin. **22**:10 (Jan. 2) 1921.

Fischer reports a syndrome which he believes has not been described previously. Oppenheim in his textbook briefly reports a similar syndrome which Fischer refers to in the discussion of his case.

The patient, a man, aged 39, who about July 1, 1920, was taken with frontal headache, a week later complained of tinnitus, and about fourteen days later of a constant smell and taste of caraway. On July 29 his condition suddenly became much worse; he developed impairment in swallowing and speech with profuse salivation. During the following three days all the symptoms became markedly exaggerated, reaching the crest on August 1. There was no history of fever, but several days prior to the appearance of the paralysis the patient complained of formication on the left side of his body, also of intense yawning.

With the development of the deglutition and speech disturbance short convulsive attacks or spasms occurred. These attacks would recur about every half to one hour, so that up to forty were observed during a day.

At the height of the disturbance the patient lost the power of deglutition and speech was impossible, only a few tones resembling moans or groans being produced. Voluntary jaw movements and respiratory movements were unimpaired. Voluntary or involuntary laughing was impossible, each attempt resulting in a rudimentary facial contortion. Ocular movements were unimpaired. There was a slight nystagmus in all directions and some paresis of the left side of the face. The facial movements were slow as were movements of the tongue which, however, showed no atrophy and could be moved in all directions. There was a good pharyngeal reflex; the uvula moved normally while movements of the soft palate were noted on stimulation, but there was no coughing. Salivation was marked.

There were no other somatic findings. The reflexes, sensibility, etc., were normal. Psychically the patient was normal. The urine was normal, the blood Wassermann reaction was negative. The convulsive attacks usually lasted from fifteen to fifty seconds. An appearance of anxiety was first noted; then the head was pulled back, the mouth opened in spasm, then pulled to the right, the eyes closed, the arms flexed at the elbows and the hands became clenched. With increasing cyanosis, there appeared rather rhythmic and synchronous opening and closing of the eyes and mouth and flexion and extension of the arms, the lower limbs not being involved. During an attack no pathologic reflexes were noted, and consciousness was retained.

By August 5 the patient appeared to have practically recovered. Examination revealed a slight weakness of the left side of the jaw. Electrical reactions of facial muscles were negative.

To summarize: There was a prodromal period of several weeks with parageusia and paresthesias of the left side of the body. Later there were left sided facial paresis and difficulty in voluntary movements of the face and tongue and complete inability to speak and phonate, to chew, swallow, laugh, yawn or cough. There were profuse salivation and peculiar convulsive attacks.

The first impression was that one had to deal with an acute bulbar palsy, but the author feels that this case does not belong in either the group of bulbar or pseudobulbar palsy. Bulbar disturbance always shows involvement of motor nuclei with atrophic flaccid palsy. In the pseudobulbar cases the affection rests on involvement of the central pyramidal tracts and is only possible when there is a double-sided lesion, and then only when the cortical centers or the pyramidal fibers are injured. There is a hypertonic palsy without atrophy; there is no voluntary movement but involuntary action remains good. The facial and tongue muscles are most effected, and the pharyngeal reflex remains present; however, impairment in deglutition may occur.

It is known that the facial muscles are prone to another form of palsy in which there is retention of voluntary innervation with loss of reflex mimic action, such as laughing and crying. This is seen frequently in thalamic lesions.

Thus there are two paths for the innervation of the facial muscles: the pyramidal tract, and the extrapyramidal tracts which connect with certain thalamic centers which have to do with the coordination of mimic action. The respiratory mechanism shows a somewhat similar condition in which the automatic center is located in the medulla oblongata, while voluntary respiratory movements are cortical. Chewing, deglutition and phonation, according to Bechterew, have both a voluntary and reflex center; the reflex centers are not localized, however.

Fischer proceeds to group his findings as follows:

1. Mimic, phonation, chewing, deglutition, yawning, coughing, may be isolated and completely impaired, even with retention of voluntary function of the entire oral muscle complex. Therefore one must consider the voluntary centers and tracts of the oral musculature different from the nerve complexes which control the reflex coordination of these acts.

2. The isolated loss of these functions demonstrates the independence of the voluntary and involuntary tracts and centers.

3. The involved elements must at some place be closely connected to assume a small focus causing the trouble, as noted in this case.

Experimental work on animals has shown that the centers involved must lie between the thalamus and medulla oblongata. It would be difficult, according to the author, to place the lesion more exactly. The fact that the left facial was slighly paretic and that the parasthesias occurred on the left side of the body would indicate that the lesion was situated in the contralateral portion of the brain, but one could not state that the lesion was unilateral or bilateral.

The presence of the convulsive attacks also tends to localization. The fact that the palsy and convulsive attacks always involved the same muscles would indicate that the same focus was responsible for both troubles. Therefore a cortical lesion is not likely. The involvement of face and arms with exclusion of legs does not speak for a cortical site. The onset of the attacks with stridor, the tonic and then clonic phase would, however, point to the cortex.

Oppenheim described a case of syphilis of the pons with facial paresis, dysarthria and dysphagia, accompanied by tonic and clonic spasms of the face and neck without loss of consciousness. At necropsy Oppenheim's diagnosis could not be substantiated. According to Nothnagel, there is a spasm center in the pons. Later Ziehen and Bechterew modified this by saying that pontile lesions could only cause a tonic spasm, and that the clonic spasms were due to secondary cortical stimulation.

The author believes that the lesion in this case was in the pons, and he states that the paresthesias as noted in the case might be ascribed to the pons.

As to the etiology, Fischer believes that he has excluded syphilis, tuberculosis, hemorrhage emboli, etc. At the time an epidemic of encephalitis was present in Prague, and the author concludes that his case was one of abortive epidemic encephalitis. He realizes the lack of postmortem findings, but in conclusion states:

1. With this syndrome (once noted by Oppenheim) one has to deal with a clinical entity. 2. It has a physiologic special function for the oral musculature. 3. This apparatus may be affected as an entity.

MOERSCH, Rochester, Minn.

[EDITOR'S NOTE.—The syndrome presented by this case is of much interest and recalls the case recorded by Dr. C. K. Mills in the *Journal of Nervous and Mental Diseases* **39**: No. 2, 1912. Unilateral mimetic paralysis with sensory symptoms, similar to those mentioned by Fischer, were present in the case of Dr. Mills. Other symptoms, however, given by the latter were wanting. In the case of Dr. Mills, the lesion was shown at necropsy to be occlusion of the anterosuperior cerebellar artery, the destruction resulting involving a portion of the cerebellum, including the superior cerebellar peduncle and also certain dorsal tracts in the pons. Fischer's case appears to be one in which these tracts were implicated, as well as some additional areas in the pons and oblongata.]

DIE ALLGEMEINE HISTOPATHOLOGISCHE CHARAKTERISIERUNG DER HEREDODEGENERATION (THE GENERAL HISTOPATHOLOGIC CHARACTERISTICS OF HEREDODEGENERATION). KARL SCHAFFER, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:193, 1920.

Heredodegenerative conditions of the nervous system are characterized by certain typical features, among which are included the familial appearance, homochronicity, homotypism, homotopism, and finally by a more or less strikingly progressive course. The classification of these disorders has always been unsatisfactory; however, the author quotes R. Bing's classification as being inclusive and yet brief. Bing divides these cases into: (1) those types in which the degenerative manifestations are predominantly motor, including the spastic and dystrophic types; (2) predominantly sensory, as in the spinal and cerebellar ataxias; (3) predominantly psychic, as in familiar idiocy; and (4) the dyskinetic types, including the myotonic, myoclonic, choreic and tremor groups.

There are innumerable transitional forms which do not permit sharp classification. According to Jendrássik, the symptom grouping is the essential feature; the origin in certain pathobiologic factors, such as heredity, familialty, consanguinity and progression, supply specific characteristics. He accordingly chose the collective appellation of heredodegeneration, considering them as being closely interrelated.

The intrinsic nature of these degenerations is quite unknown; however, the histologic findings have thus far pointed the way. The close relationship that occurs clinically would suggest a similarly close relationship along anatomic lines. Schaffer was able to exhibit the following points in support of this contention: 1. The process affects only ectodermal derivatives of the central nervous system. 2. It involves not only cell processes, but also the corresponding cell bodies. 3. It is revealed by a marked swelling, particularly of the cell body, then of the dendrites and finally of the axons.

Of the twelve cases investigated by the author, mesodermal lesions were noted in only a single instance, and in this case the lesion was circumscribed and doubtless represented an inflammatory process of exogenous origin.

Histologically, there is a swelling of the structureless hyaloplasm, resulting from the accumulation of degenerated myelinoid, protagonoid and possibly lipoid, products. In the infantile type of amaurotic idiocy, for example, the process is essentially concerned with a rapid ectodermal, that is, neuronal and neuroglial, disintegration, predetermined embryologically, since it involves the most recent phylogenetic acquisitions, and ending fatally within a year.

The important point lies in the fact that in the more chronic forms, such as the juvenile type of familial idiocy of Vogt-Spielmeyer and J. K. Walter, the process is quite similar, differing only in the extension and speed of the progress. This disease is protracted, and ends lethally only as a result of some intercurrent disease. Cerebellar heredodegenerations are essentially chronic and closely analogous. The finer changes in these include moderate swelling and simple chronic atrophy of the ganglion cells and nerve fibers, leading to a progressive cytolysis. The weblike structure of the cell body and dendrites is at first accentuated by the incrustation-like impregnations which become fragmented, first into larger particles, later into small spherical bodies, finally appearing as dust in the protoplasm. This is by no means to be identified with ordinary lipoid granules, since it is stained only with silver and therefore has been designated as "silver granular degeneration." To this process is added neuronophagia. The nucleus of the cell becomes homogeneous, deeply stained, shriveled, and ultimately melts into the cell forming a uniform mass. The picture is essentially the homologue of Nissl's chronic nerve cell degeneration. In the nerve fibers there is an irregular swelling of the myelin sheaths resulting in the formation of globules located along the course of the axons; its ability to be stained by Weigert's method is gradually lost. The axon itself shows areas of swelling, giving the appearance of a string of pearls of irregular size. This process is completed later than that affecting the myelin sheaths. The degeneration may involve only one system of neurons with their cell processes, for example, primary lateral sclerosis; or it may involve a chain of neurons, as in amyotrophic lateral sclerosis; finally, there may be such a combination as primary lateral sclerosis with degeneration of the olivocerebellar tracts. Disturbances of myelogenesis and myelonecrosis go on hand in hand. The word primary should be reserved for such degenerations as are bilaterally symmetrical, strictly parenchymatous, endogenous, and without the slightest evidence of a vascular reaction. Schaffer insists that these degenerations are invariably gangliocellular, involving first the cell, later its processes; a primary degeneration of the nerve fiber, as is assumed by Schmaus and, for instance, by Merzbacher, in aplasia axialis, has not been proved. He thinks this assumption was simply the result of the inability to detect changes in the nerve cells before modern methods came into use.

The processes noted are briefly: (1) swelling of the ganglion cells, whereby the net-like structure is thrown into relief; (2) wasting and disintegration of the Nissl granules; (3) development of hyaloplasmatic degeneration products resulting in swelling of the cell body; (4) disintegration of the perinuclear reticular structures, the so-called "silver granule degeneration"; (5) atrophy of the diseased cells, and (6) neuronophagia.

The disappearance of the Nissl granules in this process can be explained by the fact that after this material is used up the degenerated hyaloplasm cannot replenish it. Both the acute and the chronic forms have identical changes, the most characteristic of which is swelling. In an acute form, however, there is insufficient time for atrophy, which accounts for the difference in the histologic picture. The swelling itself is not specific, since it may be seen in severe infections also.

That heredodegeneration of the central nervous system is determined embryologically is proved by the fact that ectodermal structures alone are involved; also that there is a sharp selection of the structures involved, resulting in a characteristic appearance; and that phylogenetically and ontogenetically younger structures, more subject to injury, tend to be involved.

The myelodysplastic stigmas are further shown macroscopically by tectonic variabilities in the contour of the gyri of a pithecoid nature and by the occasional presence of a fetal sulcus cylindricus of His, indicating a subnormal organization; microscopically they are indicated by abnormal form and structure, such as double nuclei, abnormal localization, abnormal size and number, presence of fetal cells, abnormal slenderness of the nerve fibers, multinuclear glial formations and heterotopias.

While these diseases are both clinically and anatomically closely related, the underlying nature of them has not been determined. Such terms as deficient vitality (Jendrássik), premature senility (Bing, Higier) and wearing out of hereditarily weak tracts (Edinger) do not mean that we are any nearer the solution. The question remaining is, What influences determine the abnormal substratum? It probably can never be answered by histologic methods alone.

WOLTMAN, Rochester, Minn.

UEBER EINE EIGENARTIGE FORM VON SCHLAFSTOERUNG IM KINDESALTER ALS SPAETSCHADEN NACH ENCEPHALITIS EPIDEMICA (A PECULIAR FORM OF SLEEP DISTURBANCE IN CHILDHOOD AS A SEQUEL TO EPIDEMIC ENCEPHALITIS).

FRITZ HOFSTADT, München. med. Wchnschr. **67**:1400 (Dec. 3) 1920.

During the early months of 1920 the author had occasion to study a peculiar disturbance of sleep occurring in twenty-one children varying in age from 2 years and 3 months to 13 years. In several of the cases the condition appeared following epidemic encephalitis with chorea during the patients' residence in the hospital.

The history in all cases was quite similar. For weeks or months children who previously conducted themselves normally were unable to go to sleep before 5 or 6 a. m. During the night they presented a marked motor and psychic activity, while in the daytime they conducted themselves more or less normally. Medication and other therapy were of no avail.

The conduct of the children was somewhat as follows: At the usual bedtime they would at once begin their motor restlessness, toss themselves from side to side, sit up, arrange or change the pillows, lie down again and seem prepared to go to sleep, only again to be driven to their motor activity. Some

of the children would grimace, protrude the tongue, moving it about, or place their fingers in their mouth, wipe saliva over their face or spit. Some children would play with the bedclothes or tear up their own wearing apparel. One of the children, at the height of his motor activity, jumped out of bed yelling, threw himself on the floor, jumped up again, lay down, twisting about and grinding his teeth. Some of the children remained quiet; others talked to themselves, attempted to entertain themselves with their surroundings, sang, whistled, prayed or cried. The children were oriented in all spheres, gave prompt replies, but seemed to be hasty in making answers. They could give no reason for their motor restlessness, liked to sleep, were not fearful, had no pain or discomfort. When asked to remain quiet, they could do so for only a moment. The night was spent in this manner. Usually by 5 or 6 a. m., or sometimes even by 2 or 3 a. m., a deep, quiet sleep would come on, the patient sleeping until the following noon or sometimes only for three or four hours. During the day little sleep was obtained, though the surroundings were most agreeable. The children frequently went weeks, even months, with only from four to six or even fewer hours of sleep each night.

Most of the children appeared to be normal psychically during the day. Some, however, appeared to be dejected, shut-in, shy, while others were easily excited and irritable. Physically the little patients became pale, their eyes were sunken and there was loss of weight.

Hofstadt was able to observe the development of agrypnia in five cases of lethargic encephalitis while the patients were still in the clinic. He gives a brief history of each.

Nine other patients came to the clinic because of insomnia which had existed for weeks or months. The histories showed that the children had "grippe with subsequent sleeplessness," "grippe with choreiform movements," "brain grippe," all of which the author believes should be classified as epidemic encephalitis. No difference was noted in the severity or duration of the sleep disturbance as compared with the lethargic or choreic type of encephalitis. In the choreic form the interval between the appearance of the insomnia was shorter, or no interval was present.

Seven other patients had no history. Nothing was known regarding encephalitis, grip or other ailments. The insomnia and motor and psychic disturbance were said to have appeared without known cause. The symptoms and course were identical with those in the cases mentioned. The author believes that these children had, or at least harbored, the virus of encephalitis, presenting no apparent signs of the ailment at the time of onset, and that the disturbance of sleep was a secondary manifestation.

In this series of twenty-one cases the appearance of disturbance of sleep varied from being present with the encephalitis during the acute period up to seven weeks following apparent recovery. All the author's cases were noted between the months of February and the middle of May.

The agrypnia of encephalitis is so pronounced as to be characteristic. In none of the other childhood affections is such a profound change to be found, not even in the anxiety states which frequently cause marked insomnia. Severe forms of agrypnia are known to the psychiatrist, such as is found in pareses, arteriosclerosis, senile dementia and in the acute psychoses or collapse delirium. In many of the patients mental symptoms appeared during the early stages of the encephalitis, and the psychic activity of the patients during the agrypnia might be considered psychogenic, especially when the purposeless acts

of the child are considered. However, the absence of serious psychic or motor manifestations during the day speaks against a psychogenic basis. Furthermore, none of the patients gave a history of a neuropsychopathic constitution.

For the present the author feels that he must pass over the question of the organic changes caused by the disease in its relation to the sleep disturbance. Whether the agrypnia depends on a disturbance of a sleep center, or is due to active toxic agents of the disease, still remains a question.

Similar disturbances of sleep as sequelae of encephalitis have been reported from Heidelberg, Meran and Vienna.

In conclusion the author states that little has been found of avail in the line of medication. The usual soporifics and sedatives are useless and even tend to increase the excitement. Small doses of veronal have at times been helpful. The best results have been obtained by means of a warm dry pack, the child being wrapped in warm dry blankets which at the same time serve as a restraint. With the production of perspiration, sleep is induced and the patient rests quietly until morning.

As to prognosis, the author has little to offer, the prognosis being much the same as that for encephalitis. The majority of the patients remain in practically the same condition after a period of six or eight weeks. In only a few cases have there been any noteworthy signs of improvement.

MOERSCH, Rochester, Minn.

REPAIR OF PERIPHERAL NERVE INJURIES. G. CARL HUBER, Surg., Gynec. & Obst. **30**:464 (May) 1920.

The author's report covers the result of 279 operations in twenty-one series of experiments, from which 70,000 slides were made. The operating was done by Drs. Dean Lewis, J. F. Corbett, Bryan Stockey and T. Rosberg. The pyridin silver method for staining the tissues was generally used. Thirty-seven experiments were made in an attempt to prevent the formation of amputation neuromas. It was found that amputation neuromas were prevented by injecting 5 c.c. of absolute alcohol into the nerve, 0.5 to 0.8 cm. proximal to the point of section. Tangling or crossing of the neuraxes, which is ordinarily present in these neuromas, was absent in patients treated by alcohol injections. In twelve control experiments the sciatic nerve of rabbits was injected with absolute alcohol without section of the nerve. Paralysis followed but neuraxes grew down through the injected area and ultimate recovery of function resulted. From experimental results, injection of absolute alcohol did not seem to be deleterious.

The greater number of experiments dealt with nerve transplants. In seventeen cases of autotransplants of the superficial radial into a 3 cm. gap in the sciatic of dogs, using several segments of this nerve, the funicular structure of the transplant was maintained. Neuraxes penetrated the distal sciatic in experiments terminating longer than twenty-six days after the operation. Ultimate recovery of function of the muscles supplied by the sciatic occurred. Six experiments were made with homotransplants; the sciatic of one rabbit was resected and a segment of a suitable length taken from the sciatic of another rabbit and inserted into the gap. Four of the animals were under observation from seventeen to eighty-three days. In each of the experiments, downgrowing neuraxes had either penetrated or extended through the central end of the transplant and reached the distal sciatic, justifying the use of homoneuro transplant. Thirty-nine experiments deal with hetero-nerve-transplants. In the majority of these, one or two segments of the sciatic of a guinea-pig

were used to bridge the defect in the sciatic of a rabbit caused by reaction. The duration of the experiments ranged from three to 358 days. Serial section showed, in cases in which neuraxes were present in the distal segment, that many of the neuraxes had passed outside the transplant and into the distal stump. The author states that while regeneration through a heteronerve-transplant may be obtained experimentally, the outcome is less certain and less satisfactory and in general requires longer time than when autonerve-transplants or homonerve transplants are employed.

In experiments with degenerated nerve transplants, it was shown that regeneration is no better than when nondegenerated transplants of the same kind are used, but that degenerated autonerve transplant and homonerve transplants may be used, if opportunity presents, with assurance of success.

In experiments with homotransplants stored in petrolatum and in liquid petrolatum at 3 C. and in 50 per cent. alcohol at room temperature, return of function in the muscles supplied by the sciatic, and downgrowth of neuraxes into the distal sciatic were obtained, showing that nerves stored in these substances may be used with success as transplants. Wrapping the suture line with various material was tried out. Cargile membrane was absorbed quickly and could be of little use. When alcoholized, these membranes were not absorbed within a period of from five to six months. Sheaths of fascia lata wrapped around the transplant caused increased proliferation of connective tissue, which the author thinks may be detrimental. In six experiments prepared carotid sheaths were used to wrap the transplant. These remained in place six months and excited less connective tissue growth than fascial sheaths. They have no advantage over alcoholized Cargile membrane. In two experiments the transplant was wrapped in a fat sheath. In the one examined 238 days after operation, the fat was replaced by dense connective tissue, thus arguing against the use of a fat sheath. In thirteen experiments an arterial tubular suture by the method of Foramitti was used to bridge defects in nerves. In eleven of these experiments kept longer than four months, downgrowing neuraxes had passed through the lumen of the artery and reached the distal nerve in which beginning regeneration was noted.

The author's summary of the results of experimental work indicates that the most favorable results are to be obtained from the use of autonerve transplants, using sensory nerves in the form of cable grafts. The type of nerve, the funicular arrangements and whether the central end is placed distally or not, are not important. Accurate end to end suture, careful technic, and a dry field are essential. Fresh homonerved transplants serve as well as autotransplants if available. Experimental work justifies the use of nerve stored in petrolatum, liquid petrolatum or 50 per cent. alcohol. Sheathing about a transplant is not necessary and serves no useful purpose although not harmful. Of the various materials used for this purpose, alcoholized Cargile membrane is the best.

OTT, Rochester, Minn.

CONSIDERATIONS SUR LA FORME HYDROCEPHALIQUE MENINGITE DE LA CEREBROSPINALE DES NOURRISSONS (THE HYDROCEPHALIC FORM OF CEREBROSPINAL MENINGITIS IN INFANTS). L. CAUSSADE and A. RÉMY, Paris méd. **11**:129 (Feb. 12) 1921.

This interesting article begins with this paragraph:

"Infection of the cerebral ventricles is a serious complication of cerebrospinal meningitis. It is a frequent cause of failure of specific serotherapy. It may occur at all ages, in adults and children. However, it is of especial

interest in infants because, instead of being often clinically latent, as in adults, it has a recognizable symptomatology, and because from a physiopathologic point of view it gives information in regard to the functions of the ventricles and the neighboring cerebral nuclei. Finally, from the therapeutic point of view, the unossified state of the skull makes it possible to treat the infected ependyma by intraventricular injections of serum."

The authors review the literature briefly, recalling the various terms which have been applied to this condition: "chronic posterior basilar meningitis," "chronic cerebrospinal meningitis of the cachetic variety," "meningitic hydrocephalus," "localized cerebrospinal meningitis with ependymitis resulting in a closed cavity" and "meningitic pyocephalus." All of these terms are intended to describe a condition of cerebrospinal meningitis complicated by an acute seropurulent ependymitis, with more or less complete occlusion of the openings between the ependymal cavities and the subarachnoid spaces. The clinical syndromes resulting from this are first, ventricular hypertension, and then, hydrocephalus.

The anatomic basis of the condition is the narrowness of the openings (foramina of Monro, Magendie and Luschka) between the ventricles and the subarachnoid space in infants as compared to their size in adults. The thinnest purulent layer will suffice to block them. Clinically the condition is hard to diagnose in infants at its onset. Consequently the involvement is considerable, and walled-off foci have already formed in the arachnoid meshes before serotherapy is begun.

The authors give a case report to illustrate the symptoms of the syndrome. This emphasizes: (1) the long duration of the latent period before signs of hydrocephalus appears, (2) the masking of the condition by respiratory or gastro-intestinal symptoms or convulsions, (3) apyrexia, (4) successive stages of improvement and relapse, (5) failure to obtain abnormally large or even normal amounts of cerebrospinal fluid by lumbar puncture, (6) difficulty of injecting antimeningitic serum, (7) the presence of a lymphocytosis indicating cure in the lumbar spinal fluid combined with aggravation of the clinical picture, and (8) loss of pulsation over the large fontanel.

The authors emphasize the fact that these symptoms do not *necessarily* indicate closure of the ventricular cavities, but may be produced by obstruction of the passages from the cerebral to the spinal arachnoid spaces and that the only diagnostic signs are those directly indicative of increased tension in the ventricles. Among these are: (1) progressive increase of the volume of the skull, (2) enlargement of the fontanels, (3) spreading of the sutures, (4) softening of the skull bones (cranio-tabes), (5) pushing downward of the eyeballs, and (6) appearance of a well marked collateral venous circulation. Symptoms not mechanically due to the ventricular hypertension are: somnolence, stupor, convulsions, tetaniform rigidity and rapid emaciation.

In discussing treatment, the authors emphasized the need of intraventricular injection of serum as soon as a ventricular block is suggested by the clinical picture.

These practical suggestions are made: 1. By connecting the needle with the syringe by a piece of rubber, laceration of the brain by the needle through movement of the syringe is avoided. 2. A large amount of fluid should be withdrawn. 3. The serum to be injected should be heated to body temperature. 4. The ventricles and arachnoid spaces should be washed when possible by injecting fluid into the ventricles and allowing it to drain out through a needle inserted into the lumbar subarachnoid space. 5. Alternate the injections

between the two ventricles. 6. Injections should be made only every other day. 7. Block any dripping of fluid through the hole made by the needle, by a tampon soaked in collodion.

The reader will find an article by Walter E. Dandy, entitled "The Cause of So-Called Idiopathic Hydrocephalus," published in the *Johns Hopkins Hospital Bulletin*, March, 1921, of interest in connection with this abstract.

KRAUS, New York

A COMPARATIVE STUDY OF PERSONALITY TRAITS IN EARLY AND LATE DEMENTIA PRAECOX. CHARLES E. ROWE, State Hosp. Quart. **6**:167 (Feb.) 1921.

Of the 150 cases studied, 92 showed psychotic symptoms before the age of 30; the other group of fifty-eight cases had their mental breakdown after 30 Sixty-nine were males, of which 53 were in the group under 30 years and 16 over 30; 81 were females, 39 cases in the early group and 42 in the later. In general, the analysis was made along four lines, namely, marital condition, physical stigmas, intellectual endowment and psychic peculiarities.

As to the marital condition: Of the 92 patients under 30 years, 73 per cent. were single and 27 per cent. were married; of the 58 patients over 30, 60 per cent. were single and 40 per cent. were married.

Physical Stigmas: These include such abnormalities as weakness, small stature, youthful appearance, malformation of the cranium and of the ears, high and narrow palate, abnormal growth and distribution of hair, defective development and irregularities of the teeth. Abnormalities of this type were found in 23 per cent. of the cases, of which 18 per cent. occurred in early life and 5 per cent. in later life. The low percentage of physical abnormalities found should, the author believes, be attributed to the fact that they were not carefully recorded rather than to their absence.

Intellectual Development: The material did not lend itself readily to analysis, the patients coming as they did from locations in which educational advantages were widely varied. In making this analysis the standard followed has to a certain extent been that used by the State Department of Education, namely, that children at 14 years of age, normally bright, should be in the first year of high school, at 13 years of age in the eighth grade and at 12 years of age in the seventh grade, etc. A child two years behind this schedule is considered as making slow progress. Of the cases developing in early life 53 per cent. of the patients appear intellectually normal with 47 per cent. backward; in later life 75 per cent. were intellectually normal with 25 per cent. backward. In patients developing mental symptoms after 30, 32 per cent. of those normally intellectual were found to have had two years of high school or more and 20 per cent. were bright, but had had a limited education. Of the group in early life 20 per cent. had had two years of high school education or more and 10 per cent. normally bright, a limited educational training.

Personal Peculiarities Existing Previous to the Development of the Definite Psychic Symptoms: These are analyzed according to three types of make-up. First, the "shut-in" type which includes the shy, seclusive and the nervous, sensitive, conceited types of Kraepelin; second, the lazy, wanderer type, and third, the conscientious, docile type. In turn the shut-in type is divided into two classes, those who never have made friends and who have led a typically seclusive life and those mildly seclusive, who have had a few friends, mostly of the same sex. In this analysis of the 150 cases, I considered 48 per cent. to be definitely seclusive. This percentage is somewhat lower than that found

by Hoch. Of the fifty-eight cases developing in later life, 47 per cent. were of the total cases studied were placed in the diligent, docile type, and all of cent. were included. It would appear that this definitely seclusive type prevails almost in equal proportion no matter what the age of the person at the time of the development of his mental breakdown. In the group called mildly seclusive were placed 21 per cent. of my total cases. Here this type of make-up occurred in 29 per cent. of the cases developing in later life and in only 16.5 per cent. of the cases developing in early life.

Patients considered as socially normal made up only 11 per cent. of the total number studied, or 17 cases out of 150. Of cases of mental disorder occurring in people over 30, 19 per cent. were socially active and in the cases under 30, 6.5 per cent. As to those considered of the lazy, wanderer type, 11 per cent. were found; 5 per cent. occurred in people developing dementia praecox late and 15 per cent. in patients younger than 30. Eight per cent. of the total cases studied were placed in the diligent. docile type, and all of these cases occurred in persons who showed a psychosis in early life, or 13 per cent. of the ninety-two cases at hand.

POLLOCK, Albany.

INTERPRETATION OF MUSCLE FUNCTION IN ITS RELATION TO INJURIES OF THE PERIPHERAL NERVES. C. C. COLEMAN, Surg., Gynec. & Obst. **31**:246 (Sept.) 1920.

The report is based on material collected from the neurosurgical services of General Hospital No. 11, Cape May, N. J., and General Hospital No. 41, Staten Island, N. Y. In peripheral nerve injuries the degree of disability is determined by the loss of motor function and the presence of regeneration by return of motor function, and not by sensory loss. Motor function is easily misinterpreted, which often leads to wrong diagnoses and to the attributing of good results to operations when such are not present. In analyzing muscle function as modified or abolished by nerve lesions, the supplementary motions in the upper extremity and those possible or likely to be met with in the lower extremity, are given. In motion at the shoulder, the action of the paralyzed deltoid may be substituted rarely by the supraspinatus in conjunction with the fixation muscles of the shoulder. Flexion of the forearm in paralysis of the musculocutaneous may be performed by supplementary action of the brachioradialis. With musculocutaneous and musculospiral paralysis, the motion of the paralyzed muscles may be made possible by the action of the pronator radii tires. Extension of the forearm is seldom absent, due to the persistence of the long head of the triceps, paralysis of which is usually associated with brachial plexus injury, due to the branch to this part of the muscle being given off high up.

There is no substitution in pronation with median paralysis, but the brachioradialis brings the wrist to the neutral position but not farther. The same may be said of supination when the biceps and supinator brevis are paralyzed. Flexion of the wrist is accomplished by the action of muscles supplied by both the median and ulnar nerve, and is possible with only one of either nerves intact. With both these nerves paralyzed, flexion may be supplemented by the extensor ossei metacarpi pollicis innervated by the musculospiral nerve. Extension of the wrist is due to the action of muscles supplied by the musculospiral. However, the hand may be elevated by strongly flexing the fingers due to tension on the slightly shortened extensor tendons. This is not possible with marked hypotonia of the extensors.

Flexion and extension of the thumbs are not effectively compensated when the primary muscles for these movements are paralyzed. Adduction of the thumb is effected chiefly by the action of abducator pollicis. Complete flexion of the fingers results from the action of muscles supplied by the median and ulnar. In combined lesions of the ulnar and median, moderate flexion of the fingers is possible by extension of the wrist. This is due to the pull on the flexor tendons caused by the position of the wrist.

In high median lesions, the patient is usually able to flex the last three fingers due to the ulnar supply to the profundus. The principal disability of median injury as regards finger movements is inability to flex the index fingers. This may be effected in some cases by extension of the wrist. Extension of the first phalanx is not possible in musculospiral paralysis, but the last two phalanges can be extended through the action of the interossei. Lateral movements of the fingers are effected chiefly by the interossei. The common extensors may spread and the long flexor approximate the fingers in supplementary action. In sciatic injuries, flexion of the leg and the thigh is usually preserved, due to the semitendinous, whose main supply is given off high up and is not injured. The quadriceps action may be substituted by the tensor fascia femoris. Inversion of the ankle, normally caused by the tibialis anticus, may be performed through supplementary action of the tibialis posticus unless the foot is held at right angles. The peroneus longus may cause plantar flexion in paralysis of the calf muscles.

OTT, Rochester, Minn.

COLONY AND PAROLE CARE FOR DEPENDENTS AND DEFECTIVES. C. BERNSTEIN, State Hosp. Quart. **6**:133 (Feb.) 1921.

Out of a total population of 2,100 inmates at the Rome State School (two thirds of which are custodial cases), over one-third, 781, are being provided for outside the institution as follows:

	Males	Females	Total
In colonies	312	237	549
On parole at home	81	46	127
On parole working	49	56	105
	442	339	781

In addition to the above, ninety-seven boys and fifty-seven girls were discharged during the year after passing through colony care and training and having therein proved themselves trustworthy were promoted as indicated in the above statistical statement to parole and finally after a successful experience on parole, for an indefinite period, depending on the reactions of the individual, to a full discharge.

The boys on farm colonies and in industrial colonies are able to make an equally satisfactory showing. The boys at the Kossuth Colony this year earned $5,120.28. They were employed variously at janitor work in stores, operating elevators, delivering goods, in coal yards, caring for lawns, furnaces, shoveling snow, assisting painters, bicycle repairing, house cleaning, etc. The boys on farm colonies under the supervision of the man and wife on the farm did the farm work and were able to produce more than enough on each farm to pay all cost of operation and in addition the boys were allowed time to assist neighboring farmers at 15 to 25 cents per hour and in this way were able to earn spending money and money for the savings bank as well as for personal

clothing. This practice also serves to train and develop the boys for parole to outside farmers where they have surely been most helpful during the past summer while farm hands have been so very scarce, and thus seventy-six boys were sent out on parole in this way between April and October, 1920, and earned $1,966.47, of which $1,072.67 was expended for their current needs, and $893.80 was sent in to us and placed in the savings bank to the credit of the boys (each boy having his own individual bank account).

The boys out on parole are able to save about one-half of their earnings for the savings bank, and the girls about twenty-five per cent. (There seems to be a greater tendency to "doll up" the girls more than the boys.)

Forty girls placed out on parole during the year earned $3,138.92, of which $2,111.74 was expended by the girls for current personal expenses, clothing, etc., and $1,027.18 was saved and placed in the savings bank to the individual credit of the girls. Fourteen of these girls who had been out nearly the whole year had earned over $150 each.

There are at least two very good arguments in support of colony care for those who are subject to or in need of assistance during the period of their incapacity or enforced limited freedom and action, namely:

First: the great need for making the limited funds available reach the largest number posible, and second, rendering the enforced restriction of freedom and economic and social limitations as humane and as little humiliating as possible.

It is our experience that the unit of from sixteen to twenty-four girls or boys living in a colony under the supervision of a man and wife, or girls under a woman and an assistant, can easily earn sufficient to support the unit and in prosperous times have a little surplus for individual savings, and in this way self-respect is engendered in the individual rather than dependency, humiliation and discouragement. Rather than handing out alms we are supervising self-earned support and instructing our patients not only in hygiene and animal inhibition, but also in habits of industry and thrift and honorable self-support, the sheet anchors of moral prophylaxis.

POLLOCK, Albany.

DE QUELQUES TRAITMENTS DE LA MIGRAINE (CERTAIN METHODS OF TREATING MIGRAINE). P. PAGNIEZ, Presse méd. **29**:45 (Jan. 15) 1921.

This paper discusses the comparative merits of the theories and methods of Bouché and Hustin (*Bull. de l'Acad. Royale de méd. de Belgique*, 4th Series **30**:723 [June 26] 1920), of Widal, Abrami, and Iancovesco (*Presse méd.* **28**: 893 [Dec. 11] 1920, and of Didsbury (*Progrès méd.* **35**:429 [Oct. 2] 1920).

Bouché and Hustin have found in migraine a group of complicated blood vascular symptoms (not enumerated here) that differ from the "hemoclastic crisis" (*crise hémoclasique* of Widal). These they consider to be of anaphylactic nature. They also claim that when an organism is sensitized to several substances, it no longer reacts to the injection of either of these substances, if it has just survived a shock caused by one of them; hence, if horse serum or crotalin be given in a series of injections, there will be less reaction to whatever substances are causing the migraine.

Reactions to these injections are generally local, but frequently severe, causing a rather extensive patch of erythema which may last a week. Injections are given weekly for two months, then fortnightly for from two or three months. One course may effect a permanent cure of the migraine, or a second may be

required in the following year or two. If horse serum has been used in the first series, crotalin may be substituted in the second, or vice versa. One injection consists of 1 c.c. horse serum or 0.1-0.2 mg. crotalin.

The mechanism of this therapy is not clearly understood. It should be reserved for cases proved refractory to other treatments. Marked disadvantages appear in the intensity of reactive phenomena that may occur, in the possible phase of aggravation, and in the chance of producing serious results hitherto unreported.

A safer and well recognized treatment consists in giving peptone (about 0.5 gm.) one hour before meals during the intervals between seizures. The resulting postponement or amelioration of attacks has often given brilliant promise at first, but end-results have been less satisfactory. Some disagreement exists as to whether the peptone merely alleviates disordered digestion, or whether it acts as a specific against this particular anaphylaxis.

Widal and his collaborators find insufficiently digested albuminoids carried in the portal vein after digestion, and stopped by the normal liver. In alimentary anaphylaxis the liver fails to function in this manner, and the phenomena of a hemoclastic crisis (sudden arterial hypotension and leukopenia) takes place. The same crisis can be produced experimentally by direct injection of these albuminoids into the vena cava. Migrainous seizures are accompanied by hemoclastic crises.

Didsbury's method is based on a conception of migraine as a chronic headache of superficial local origin. He believes that hypersensitive points and areas can always be found over the head during the intervals between attacks. These are to be massaged for thirty minutes daily. Some benefit is expected after a fortnight, and a permanent cure after continued treatments. Pagniez and others admit the existence of such cases, possibly amenable to that treatment, but contend that they are not cases of migraine.

HUDDLESON, New York.

ETUDE DE LA SUEUR DANS LES BLESSURES DE LA MOELLE (A STUDY OF THE SWEAT IN WOUNDS OF THE SPINAL CORD).

ANDRÉ-THOMAS, L'Encéphale **15**:233 (April 10) 1920.

This is a most important paper bearing on the uses of the knowledge of the relations of the voluntary to the involuntary nervous systems. The author begins his article of twenty-one pages by emphasizing the frequency of the occurrence of changes in sweating, pilomotor reactions and other sympathicogenic phenomena in tramatic lesions of the spinal cord. He has already contributed articles on these subjects.[1]

He emphasizes the difficulties—the variability of the sweating in different persons and in one person at different times, as well as the influence of ordinary psychic and physical changes. André-Thomas did not care to subject severely wounded soldiers to the discomfort of a pilocarpin test, and limited his observations to cases complicated by fever or infection.

The observations were made by inspection and touch, neither litmus nor silver nitrate paper being used.

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1. Reactions ansérines ou pilomotrices dans les lésions et les blessures du système nerveux, Paris méd., Juillet, 1918. Contribution a l'étude des centres pilomoteurs de la moelle épiniere chez l'homme, Soc. de Neurol., Dec. 5, 1918. Les réactions pilomotrices et les réflexes pilomoteurs dans les blessures de la moelle. Réflexe encéphalique et reflexe spinal. Soc. de Biol., March 29, 1919.

Two stages exist in spinal cord injuries. The first, that of shock or inhibition. In this stage sweating is absent in the paralyzed portions, or nearly absent, though the line of anesthesia and that of absence of sweating are not always the same. Sweating is only present in segments innervated by the sympathetic nerves above the lesion, that is, those which have maintained their connections with the brain. For this reason, André-Thomas speaks of sweating of brain origin (sueur encéphalique).

After the first period of shock or inhibition, a second period of overactivity of the segments below the lesion begins. With this there also occur changes in the nature of the sweating—sweating of spinal origin (sueur spinale).

The author presents a number of case reports, many with necropsy findings. He says, "The most important point to study is the level of sweating, the lower level of brain sweating, the upper level of spinal sweating, on account of the information which this sign furnishes us as to the localization of sweating centers in the sympathetic column of the spinal cord and in the diagnosis of the level of the lesion." He correlates the level of the sweat centers (involuntary nervous system) in the spinal cord segments with the level of the sensory changes (voluntary nervous system). He shows that these levels are not the same in the skin, as would readily be expected by analogy to the work of Langley on the cat. He recalls the work of Head and Riddock, published in *Brain* in 1918.

He states that the sweat centers for the head, neck and the upper part of the thorax are in segments C 8 to D 3, inclusive, and that the centers for the upper extremities lie in D 5, 6 and 7 and probably lower.

The article is worth reading in full by all who wish to better their knowledge of this method of diagnosing spinal lesions.

(The reader may refer to Gaskell's "The Involuntary Nervous System," page 38, or to the *Journal of Comparative Anatomy* **29**:424, 1918, for the anatomic basis.)

KRAUS, New York.

A PERSONALITY STUDY OF LATE DEMENTIA PRAECOX. ANNE E. PERKINS, State Hosp. Quart. **6**:174 (Feb.) 1921.

In studying the make-up and personality of thirty-eight women now in the hospital, who developed dementia praecox in the fourth and fifth decades, certain points were noticed: (1) good preservation of the personality, (2) natural affect, (3) little progression of deterioration, and (4) unnatural attitude toward marriage, with failure of adaptation to its demands. Of the entire group, thirty-three belong to the paranoid type; in these the chief characteristics may be summed up thus:

1. Twelve show remarkable preservation of the personality. They are neat in habits and appearance, industrious, more in contact with the world than the usual patient with dementia praecox and take an interest in outside affairs. Ten have never evidenced hallucinations; no mannerisms, silly laughter or masturbation have been observed, and the shallowness customary in dementia praecox is absent. Several have excellent judgment in buying, planning financially, saving money, etc., and two are not infrequently consulted about such practical matters as purchasing dress materials and designing dresses.

2. Fourteen show good emotional responsiveness, it being possible to establish with them a closer affective rapport than is commonly experienced in dementia praecox; there is less evidence of discrepancy between their ideas and mood. They are more natural, open, companionable and agreeable than patients with other types of dementia praecox.

Twelve have marked scolding reactions owing to paranoid delusions. Only three evidence depression, and at times shed tears because they wish to be at home, to earn money or to care for the family.

3. An outstanding feature is the tendency of patients with paranoid cases to continue at a certain level, with little or no progression of deterioration. Only two have undergone much deterioration. Like Kraepelin's paraphrenic types, several "do not show the deterioration in conduct or the emotional indifference so characteristic of dementia praecox. There is not the same disruption of personality because there is far less disturbance in the emotional and volitional sides of the mental life. The clinical pictures are characterized chiefly by the marked paranoid delusions. The behavior of these patients also remains well in harmony with the ideas and moods." Two might perhaps be classed as Kraepelin's confabulatory type, having delusions of royal birth, disowning relationship to parents, considering themselves queens, princesses and the bride of Christ. Two might be called the fantastic type, having a medley of extremely absurd, disconnected, bizarre delusions.

4. The unnatural attitude of the dementia praecox patient toward marriage is shown by the majority. Of the sixteen who never married, several repeatedly broke engagements, shrinking from approaching marriage; others showed marked indifference toward men. In some cases adequate history could not be obtained.

Of the twenty-two who married, nine separated from their husbands, and at least five others are known to have failed to adapt themselves successfully to marriage. Six developed the psychosis after childbirth; one left her husband shortly after the birth of her child; another definitely broke down after the birth of her second child—in the early forties. Dementia praecox often develops during the puerperal period when a woman has found adaptation difficult, for once she has borne a child, the bonds are tighter, she cannot so easily get away, hence develops a psychosis after or near childbirth. One insisted that her marriage had been annulled, always signing her maiden name and addressing her children's letters with this name. One developed a definite psychosis after an attempted sexual assault.

POLLOCK, Albany.

APHASIE DE WERNICKE—UNIQUE SEQUELLE D'UNE CONTUSION CEREBRALE PAR CONTRE-COUP (WERNICKE'S APHASIA—AN UNUSUAL SEQUELA TO CEREBRAL CONTUSION BY CONTRE-COUP). F. BREMER, Rev. Neurol. **27**:132 (Feb.) 1920.

The clinical picture in Bremer's case may be detailed as follows: The patient, a mason, aged 38, fell 21 feet from a scaffold, striking directly on his head and sustaining a slight scalp laceration in the midparietal region on the right, 6 cm. from the sagittal suture. When first seen by the author, twenty-four hours after the accident, the patient, although unconscious, displayed great motor restlessness associated with continuous but utterly senseless speech. Respiration was normal, the pulse rate 85, and the temperature 38.5 C. No pupillary disturbance was noted, nor could there be determined evidence indicative of basal fracture, paralysis, cerebral compression or meningitis.

No essential change was marked until the fifth day when the motor agitation was noted to have subsided considerably and sensibility to have reattained its normal status. Response to interrogation at this time, was definitely paraphasic. On the seventh day, the patient, although still bed-ridden, was observed to smile and extend his hand when approached. It was impossible, however, to secure comprehension to spoken words and replies were still paraphasic.

Lumbar puncture performed on the tenth day after the injury, showed xanthochromia and 50 cells (60 per cent. polymorphonuclears), with increased albumin but no disturbance in the chlorid content, indicating aseptic reaction to intrameningeal hemorrhage, presumably of slight degree.

Neurologic examination made on the fifteenth day, by which time the patient was entirely ambulant, revealed slight paresis of the right arm, unassociated with incoordination or adiadokokinesis. No change was noted in sensibility and, except for slight exaggeration of the wrist reflex on the right, no disturbance could be elicited in the deep or superficial reflex response. Vision was apparently normal.

Mimetic power and articulation showed no defect, but there was absolute lack of comprehension for spoken words and inability to name presented objects. Spontaneous and repetitional speech were characteristically paraphasic with evidence of periphrasis and perseveration. There was no evidence, however, of alexia or agraphia. Memory showed lacunar amnesia covering the accident and subsequent shock-period.

After a month, there was noted some improvement in the comprehension of spoken words and disappearance of the pleocytosis and the wrist jerk inequality. Roentgenologic examination of the head at this time proved completely negative and, aside from headache, there was absence of subjective somatic complaint.

Pathologically, Bremer suggests the possible dependence of this syndrome on focal hemorrhage or softening in the left cerebral cortex.

RAPHAEL, Kalamazoo, Mich.

THE REPRODUCTIVE GLANDS AND MENTAL DISORDER, WITH SPECIAL REFERENCE TO DEMENTIA PRAECOX. GEORGE H. KIRBY and CHARLES E. GIBBS, State Hosp. Quart. **6**:147 (Feb.) 1921.

We recently made an attempt to determine whether or not by simple physical examination of the male genitals any gross differences could be determined between dementia praecox and other psychoses. Within a few days, 313 patients were examined. The psychoses were: dementia praecox, 186; manic-depressive, 50; general paralysis, 49, and a miscellaneous group of 28. Most of the patients were taken alphabetically in several mixed wards, the diagnosis being unknown to the examiner. Only what seemed to be a definite departure from normal limits was recorded as abnormal. The testes were palpated for size and consistency.

As shown by the tabulation the dementia praecox patients showed the highest rate of variation in size, although the differences were not great. Patients with general paralysis and dementia praecox showed a definitely higher rate of changes in consistency, chiefly softening, than did patients with manic-depressive psychosis. This difference was also definite in cases in which there were changes in size and consistency in the same person.

The softening in general paralysis was more marked than in dementia praecox. These differences perhaps become more significant in the light of the age distribution, since 76 per cent. of the patients with dementia praecox were under 41 years of age, while only 28 per cent. of patients of manic-depressive psychosis and 29 per cent. with general paralysis were under 41.

The variations in size and consistency in dementia praecox were greatest at the age extremes, 100 per cent. of those under 21 and 83 per cent. of those over 41 showing some variation as to size or consistency. Although only 24

TABLE 1.—Variations in Size and Consistency of the Testes

	Number of Cases	Abnormal Size, Per Cent.	Abnormal Consistency, Per Cent.	Abnormal Size and Consistency, Per Cent.
Dementia praecox	186	56	47	31
Manic-depressive psychosis	50	46	22	12
General paralysis	49	42	58	28
	285			

Columns showing abnormal size and abnormal consistency include cases showing both changes in same person as shown in the last column.

TABLE 2.—Age Distribution

	Under 41 Per cent.
Dementia praecox	76
Manic-depressive psychosis	28
General paralysis	29

per cent. of patients with general paralysis were over 50, the impression was gained that the softening was more like that found in senile cases than was the softening in dementia praecox.

It is appreciated that this is a gross method, that some of the cases may be within normal limits, and that other factors, such as circulation and nutrition, may have a bearing.

Various other deficiencies of general development were observed in a significant number of cases, but no great differences in the three groups were noted, except that the penis was considered to be small or short in 59 per cent. of the cases of dementia praecox and in 40 per cent. of the cases of general paralysis, but in only 28 per cent. of the manic-depressive patients.

Pollock, Albany.

CLASSIFICATION OF GOITER: AN ANALYSIS OF ONE HUNDRED CASES. Carrington Williams, Am. J. Med. Sc. **161**:223 (Feb.) 1921.

The author's material was the patients admitted to the surgical clinic of St. Luke's Hospital of Richmond, Va. He divides the cases into three groups. Group 1 contains the cases of simple goiter. In this group there were sixty-one patients of whom fifty-seven were females and four males. The duration of the goiter varied from two months to twenty-five years with an average of seven and one-half years. This group of patients had practically no symptoms except those of pressure.

Group 2 presented an interesting picture. The patients were middle-aged and had had goiter for a long time, with no symptoms. The goiter suddenly began to grow, and the patients became restless and nervous and had symptoms of hyperthyroidism, but did not have a tremor or exophthalmos. There were twenty patients in this group, of whom nineteen were females. The age incidence was 34 to 58 years with an average of 47 years. The duration was from two months to thirty years with an average of eleven years. Seventy-seven per cent. of the cases gave a history of recent growth. The pathologic condition in this group was that of the simple goiter class.

Group 3 embraced the patients with the classic symptoms of exophthalmic goiter. There were nineteen patients in this group, all females. The age incidence was from 15 to 51 years with an average of 33 years. The duration

of symptoms was from two months to seven years, with an average of two years. Fifty-seven per cent. of these cases gave a history of a recent growth of the goiter. The pathology in this group was hyperplastic parenchymatous goiter which is composed of closely packed tubules lined with multiple rows of large cells with little or no colloid material. Williams believes that surgery is the cure for all the groups. He says that roentgen-ray and radium treatment should be limited to group 3, and that it is conceivable that in the future the radiologist may take preference over the surgeon in the treatment of group 3.

WILSON, Philadelphia.

LE MECANISME DU HOQUET (THE MECHANISM OF HICCUP). H. ROGER and E. SCHULMANN, Presse méd. **29**:161 (Feb. 26) 1921.

Graphic records were made of one of the "epidemic hiccup" cases, discovering several interesting features not ordinarily observed clinically. The subject was in the third day of the disease; typical hiccup occurred at every third or fourth breath, and caused vomiting on ingestion of any substance. His general condition was good in spite of fatigue, hunger and insomia. A remission of the hiccup took place for several hours during the investigation.

The apparatus was set to show inspiration on the ascending stroke. The curves (illustrated in the article) showed that each characteristic spasmodic inspiration was immediately preceded by a quick forced expiration, unnoticed clinically. The expiratory spasm almost always began at the end of inspiration, but sometimes during the expiratory pause or the beginning of inspiration.

Abortive spasms were not uncommon; occasionally the inspiratory spasm was lacking, exceptionally the expiratory. Both abortive and complete spasms were reduplicated at times.

During the period of hiccuping, respirations were irregular and unequal, with an amplitude from one-third to two-thirds of normal, and a rate about one and one-half times normal. During the period of remission, some abnormalities still persisted, for example, inspiration was longer, and the expiratory pause shorter, than normal, and one abortive hiccup was registered.

Fluoroscopy in the myoclonic phase (period of hiccuping) showed a transitory tetanizing of the diaphragmatic arches. Instead of contracting normally they were agitated by fibrillary twitchings. No attempt was made in this paper to theorize on the paths of the nervous impulses involved.

HUDDLESON, New York.

HEMIHYPERTROPHIE CONGENITALE DES MEMBRES ET DU PAVILLON DE L'OREILLE CORRESPONDANT (CONGENITAL HEMIHYPERTROPHY OF THE EXTREMITIES AND IPSOLATERAL PINNA). C. ROUBIER, Rev. Neurol. **27**:147 (Feb.) 1920.

Roubier directs attention to the ubiquity of the minor or so-called physiologic asymmetries of limb size and contour in contradistinction to the distinctly infrequent occurrence of true congenital hemihypertrophy. The latter may be of three types: (1) complete, involving an entire half of the body or at least the arm and leg, (2) partial, involving a single limb, and (3) facial, involving one side of the face. Of these three types, the first, of which the author's case is an example, is perhaps the most uncommon, not more than forty such cases having been reported to date in the extensive literature available on this subject.

The case presented by Roubier was that of a single, young adult soldier with apparently negative family and personal history except for bodily asymmetry dating from birth. Examination revealed the presence of definite hemihypertrophy (including both skeletal and soft tissues) which involved both extremities on the right and the corresponding pinna, but apparently did not affect the facial elements or the trunk. There was no integumentary abnormality on either side or evidence of adiposis. The superficial veins, however, were quite prominent, particularly on the right, and there was additionally noted, on this side, slight cyanosis of both extremities with edema extending over the leg and ankle. The temperature also, over this leg, was somewhat higher than over the left. No neurologic disturbance could be detected except the apparent diminution of muscular force on the right. Examination of the special sense organs and viscera was completely negative, and it was equally impossible to elicit evidence suggestive of psychic disorder.

Roubier considers the various hypotheses which have been advanced as to the etiology of this condition, commenting briefly on the possibility of congenital hemilateral vasomotor imbalance or paresis, of venous obstruction dependent on fetal malposition, and of fundamental metameric disturbance or faulty mesodermic development during embryonal life, but concludes, finally, that none of these conjectures are validly tenable and that the actual causative mechanism must still be regarded as preeminently obscure.

The article is illustrated with three excellent figures, and there is appended an extremely comprehensive bibliography.

RAPHAEL, Kalamazoo, Mich.

EPILESSIA, ANAFILASSI E DISTIROIDISMO (EPILEPSY, ANAPHYLAXIS, AND DYSTHYROIDISM. V. M. BUSCAMO, Schweiz. Arch. f. Neurol. & Psychiat. **7**:2-304, 1921.

The author calls attention to his publication, in 1915, in which he first formulated the theory that idiopathic epilepsy is nothing more than an anaphylactic crisis provoked by the presence in the blood of certain abnormal proteins of thyroid origin. He also assumed that the so-called symptomatic epilepsies, e.g., those of arteriosclerosis, general paralysis of the insane, had the same origin, basing his theory at that time on a certain number of cases studied clinically and anatomo-pathologically in which he always found an abnormal protein crystalline in form in the thyroid colloid, coincidently with lymphocytosis, etc. Since his first article no proof has been brought against his theory, but many authors, of whom he cites twenty or more, have reported facts corroborating his theory, some experimental and others clinical.

In proof of his theory he now presents a total of 100 cases in which the thyroid was examined with care. Of these patients, thirty-nine were epileptics or persons having epileptiform seizures and the other sixty-one were not epileptic or suffering from epileptiform seizures. He offers this interesting result: The protein crystals were present in the thyroids of 84 per cent. of the epileptics or those suffering from epileptiform seizures and in 40 per cent. of the thyroids of nonepileptics and persons not suffering from epileptiform seizures. He suggests as treatment for epilepsy partial thyroidectomy, with successive administrations of thyroid extract, in order to eliminate the production of the abnormal protein. He mentions Crile's removal of one suprarenal and three-fourths of the thyroid (also section of the cervical sympathetic) as analogous to his suggestion for treatment although arrived at from another point of view. A full bibliography is appended.

GURD, Ann Arbor, Mich.

A SKETCH OF THE ORIGIN OF THE CEREBRAL HEMISPHERES.

C. JUDSON HERRICK, J. Comp. Neurol. **32**:429 (Feb. 15) 1921.

The endbrain (telencephalon) of lower vertebrates is dominated by the olfactory system, and the differentiation of this region in all higher forms appears to have taken place largely under the influence of various systems of nonolfactory fibers which have grown forward into this olfactory territory. Increasingly complex correlations of the various other senses with smell have involved the elaboration of separate correlation centers in the forebrain for each of these reflex patterns, different in each species of animal according to its mode of life. The diverse patterns of cerebral architecture exhibited in the vertebrate series are, therefore, structural expressions of these functional relationships, namely, the various parts played by the olfactory and the different nonolfactory systems. Illustrations are given of the way in which this principle works out in the lower vertebrates in shaping the form of the cerebral hemisphere, and the attempt is made to reconstruct the probable history of these form changes in early Devonian times when the amphibia with fully formed cerebral hemispheres arose from ganoid fishes with more primitive brain structure, and the functional factors which operated in effecting these morphologic changes.

HERRICK, Chicago.

RECHERCHES SUR LA VALEUR DE L'ELECTRODIAGNOSTIC DANS LE DIAGNOSTIC DE LA TETANIE DE L'ADULTE (RESEARCH REGARDING THE VALUE OF ELECTRODIAGNOSIS IN TETANY OF THE ADULT).

F. NAVILLE, Ann. de méd. **8**:280 (Oct.) 1920.

The author carried out electrical reactions on three groups of cases, twelve of tetany of assured diagnosis, twelve doubtful cases and twenty of conditions clearly different in nature. He found the ulnar nerve most advantageous for these tests and recommends its continued use for the sake of standardization. The findings reaffirm the state of electrical hyperexcitability in tetany.

In an adult with tetany, either manifest or latent, a muscle twitch response is elicited by the negative closing current (negative pole on the ulnar nerve) with less than 1 milliampere; by the positive closing with less than 2 milliamperes; by the negative opening and by the positive opening by less than 5 milliamperes. A state of tetanization of the muscles supplied by the nerve is reached in the tetany case by a negative closing current under 5 milliamperes and by a positive closing current under 10 milliamperes.

In the cases without tetany, the negative closing reading was almost always above 1 milliampere, the positive closing above 2 milliamperes, the negative closing tetanization threshold always above 5 milliamperes.

The negative closing tetanization response with less than 5 milliamperes and a negative opening muscle twitch under 5 milliamperes are conclusive proof of tetany. The other alterations are suggestive but not pathognomonic of the disease.

DAVIS, New York.

THE CURABILITY OF TUBERCULOUS MENINGITIS.

FRANCIS HARBITZ, Am. J. Med. Sc. **161**:212 (Feb.) 1921.

The author calls attention to the curability of tuberculosis in other parts of the body, namely: the lungs, bones, glands, kidneys and serous membranes. There is one tuberculous condition that is still considered hopeless, namely, tuberculous meningitis and tuberculosis in the central nervous system. Harbitz thinks we should review our ideas on the subject, and says that as long

ago as 1858 Rilliet wrote about the healing of tuberculous meningitis. In 1914, Bokay collected twenty-nine cases, in all of which the tubercle bacillus was demonstrated in the spinal fluid or by the inoculation of guinea-pigs. To date there have been about forty cases of tuberculous meningitis with recovery. Harbitz reports a case in which at postmortem there were found evidences of a previous tuberculous meningitis and encephalitis with numerous tubercles on the surface of the brain as well as in the interior of the ventricles; section of the spinal cord showed areas of lymphocytic infiltration in the leptomeninges with here and there, particularly in the lumbar cord, distinct tubercles which were in the process of healing.

WILSON, Philadelphia.

ETUDE HISTO-PATHOLOGIQUE DES CENTRES NERVEUX DANS TROIS CAS DE MALADIE DE FRIEDREICH (HISTOPATHOLOGIC STUDY OF THE NEURAXES IN THREE CASES OF FRIEDREICH'S ATAXIA). G. MARINESCO and C. TRETIAKOFF, Rev. Neurol. **27**:113 (Feb.) 1920.

Marinesco and Tretiakoff, in their cases, found pathologic changes in both the white and gray matter of the cord. The changes in the white matter, particularly marked in the dorsal region, were essentially atrophic in nature, showing involvement of the posterior columns (including both endogenous and exogenous fibers) and the pyramidal tracts, as well as the bundles of Flechsig and Gowers. As regards alteration in the gray substance, there was noted, characteristically, evidence of slowly progressive simple atrophy in the cell bodies and fibers of the columns of Clarke, the posterior horns, and the posterior root ganglions. In the latter, there was additionally remarked traces of regenerative effort in the form of pericellular and, somewhat less frequently, periaxional plexi. The authors feel the gray matter disturbance to be basically dependent on primary changes occurring in the white substance, i. e., the process is essentially cellulipetal rather than cellulifugal.

No trace was detected in the cord of actual cellular disintegration or infiltrative reaction although a certain degree of apparent inflammatory reaction was determined in the cerebral and cerebellar cortices, which the authors regard as more or less incidental and wholly secondary to the process in the cord.

Comment is made on the characteristic reduction in gross volume noted in the cord, medulla and cerebellum which seems to stand in direct relation to the age of onset and the clinical duration of the disease, thus indicating, in the authors' conception, its basically developmental nature and its probable dependence on alteration (biochemical rather than inflammatory) in the myelin and neuraxial material of the centripetal neurons and ascending fiber tracts.

RAPHAEL, Kalamazoo, Mich.

SUR LA PRESSION DU LIQUIDE CEPHALO-RACHIDIEN (SPINAL FLUID PRESSURE). J. A. BARRÉ and R. SCHRAPF, Bull méd. **35**:63 (Jan. 19 and 22) 1921.

The writers conclude that the maximal normal pressure of the spinal fluid, when measured with the Claude manometer, is 20 cm. (of water) for the lying position and 40 cm. for the seated position with head lowered. They consider as pathologic any pressure exceeding 25 cm. in the first position and 45 cm. in the second. The lifting of the head in the seated position raised the pressure from 5 to 10 points.

To prevent agitation and its resultant effects on pulse and respiration the writers follow the custom of giving a hypodermic injection of sedol ten minutes before the puncture and using ethyl chlorid as a local anesthetic.

They believe that occasionally in spite of the needle being properly within the subarachnoid space, the fluid does not flow due to certain sufficiently vague circulatory alterations induced by fright and other emotions. Reassurances or the inhalation of a stimulant, such as ether, in such instances initiates the flow.

The absence of a spinal fluid "pulse" synchronous with the arterial pulsations is probably indicative of spinal fluid hypertension.

DAVIS, New York.

PARALYSIE DU NERF SPINAL PAR FRACTURE DE LA BASE DU CRANE (PARALYSIS OF THE SPINAL ACCESSORY NERVE FOLLOWING BASAL SKULL FRACTURE). G. L. REGARD, Rev. Neurol. **27**:141 (Feb.) 1920.

Regard's patient was an artilleryman, aged 38, who had fallen from a loft. When first seen, several hours after the accident, the patient was deeply comatose and examination, at this time, revealed complete areflexia, with the exception of greatly diminished knee jerks, profound depression in the sensorimotor field, myosis, marked facial pallor, frigidity of the extremities and bradycardia (60). Although it was impossible to determine primary evidence of cranial injury or depression, there was noted copious bleeding from both ears (particularly the left) and from the nose. Prosopoplegia was present on the left and lumbar puncture yielded blood-stained fluid. A small abrasion was noted on one thumb.

By the eighth day sufficient improvement had been made to permit a more exact neurologic examination. The original reflex and sensorimotor disturbance was now no longer in evidence but there was definite persistence of the left facial paralysis (subgeniculate type) with complete loss of function in the left ear. It was apparent, also, that the spinal accessory nerve on this side had suffered total paralysis, as shown by the raucous, nasalized voice, paralysis of the left palate and left sterno-cleido-mastoid and trapezius palsies.

Tetanus developed on the thirteenth day and terminated fatally six days later, the infection entry having presumably taken place through the abrasion on the thumb. Necropsy was not performed.

On the basis of the picture presented clinically, the author feels justified in the assumption of basal skull fracture, specifically of the petrous portion of the left temporal bone of the posterior longitudinal type.

RAPHAEL, Kalamazoo, Mich.

LE TRAITEMENT DE L'ENCEPHALITE LETHARGIQUE (TREATMENT OF LETHARGIC ENCEPHALITIS). L. CHEINISSE, Presse méd. **29**:146 (Feb. 19) 1921.

"Therapeutic nihilism" is discarded in favor of a method originated by Pic and approved by Netter (Traitement de l'encéphalite lethargique, *Bulletin de l'Academie de Médecine*, March 30, 1920). Most other methods that gained some repute are disapproved. There is evidence that the serum of convalescent cases is harmful rather than beneficial. Autoserotherapy has not proved valuable.

In the recommended method, 1 to 2 c.c. (adult dose) of oil of turpentine, preferably old, is injected into the cellular tissue of the abdominal wall. An

abscess should form, and is to be opened, when fluctuation is established. If no abscess develops after the first injection, a second is given two days later, and a third after another two days' interval if required. Urotropin by mouth or intravenously is also recommended as a supplementary medication but not as a substitute. The abscess treatment should be started as soon as the diagnosis is certain. Urotropin may be given later.

HUDDLESON, New York.

GERODERMIE CHEZ UN ENFANT (GERODERMIA IN A CHILD). P. HAUSHALTER, Rev. Neurol. **27**:15 (Jan.) 1920.

Report is made of a case (a boy, aged 3) in which the onset was traced to a severe attack of pneumonia at the age of 1. Otherwise, the personal and family history were negative, except for the occurrence of goiter in the mother.

Clinically, the gross picture was typical of gerodermia. In addition to the characteristic senile expression and integumentary change, however, there was noted prominence of the parietal bosces, "Gothic" palate, elongated pinnae, orchitic hypoplasia and low-pitched voice. There was no indication of dwarfism, the body length being 92.7 cm. Mentally, except for a certain gravity of manner and seriousness of interests, there was apparently no determinable evidence of deviation.

Haushalter calls attention to the fact that in most of the cases described in the literature, the condition seems to have occurred in older persons, and to have been additionally characterized by dwarfism, alopecia and often by pronounced asthenia. The probability of the existence of causative endocrine imbalance in these cases is indicated, also the desirability of their inclusion under the collective clinical designaton, genito-glandular dystrophy, as recently suggested by Souza and Castro.

RAPHAEL, Kalamazoo, Mich.

CORTICAL WORD BLINDNESS. HUBERT S. HOWE, Neurol. Bull. **3**:64 (Feb.) 1921.

The author reports the case of a man aged 55. After a peculiar attack of a feeling of being smothered, which lasted several hours, he suddenly found that he could not read his newspaper. He was not unconscious, and there were no other complaints. General physical and neurologic examinations were practically negative. His vision was 20/40 in both eyes, and the fields were normal, showing no hemianopsia. His speech and intelligence were not impaired. He could read single letters fairly well; he could copy; but he could not read whole words or sentences or write spontaneously. The author considers this case as showing pure cortical word blindness, and places the lesion in the angular gyrus. The case was exceptionally well studied clinically, and apparently represents a rare instance of a dissociated defect due to a circumscribed lesion, and as such is worthy of record.

WECHSLER, New York.

L'ESCARRE ET L'ATROPHIE CEREBRALE DES AFFAIBLIS INTELLECTUELS (TROPHIC SORES AND CEREBRAL ATROPHY IN THOSE WITH MENTAL DISORDERS). HENRI DANAYE, L'Encéphale **15**:681 (Dec. 10) 1920.

This author calls attention to the coincidence of trophic sores (bedsores) with changes in the brain in cases of psychosis which are plainly of toxi-infectious origin and associated with demonstrable pathologic changes in the brain itself. On the other hand, patients with psychosis unassociated with demon-

strable pathologic changes do not suffer from trophic sores. Of these, examples are dementia praecox vera and epilepsy. Of the conditions showing organic change, those which most often show complicating trophic sores are general paresis and senile dementia. In both of these profound anatomic changes of the brain are present.

KRAUS, New York.

THE NERVOUS SYSTEM OF A TWO-HEADED PIG EMBRYO. MABEL BISHOP, J. Comp. Neurol. **32**:379 (Feb. 15) 1921.

This is a detailed anatomic description of the brain and nerves of a laterally symmetrical dicephalous pig embryo of 22 mm. The lateral sides of the brain and peripheral nerves are normal; the conjoined medial sides show various degrees of duplicity from completely separate and normal relations of the snouts to complete union into a single individual in the trunk region. A study is made of the regulatory phenomena in the conjoined regions, and all of the anatomic evidence points toward a merely regulatory adjustment in a healthy, orderly developed, though teratologic, embryo, for wherever normality or near-normality can assert itself, it is anatomically realized or approximated. The paper is illustrated by twenty illustrations of wax reconstructions and microscopic sections.

HERRICK, Chicago.

CRANIAL DEVELOPMENT FOLLOWING ENUCLEATION IN EARLY YOUTH—POSSIBLE EFFECT IN ADULT LIFE. GELFORD DICKINSON, Am. J. Ophth. **4**:270 (April) 1921.

In the case cited, in early enucleation of the eye, the boney structure of the orbit continues to grow—especially in those parts that afford the most protection normally, the superior and temporal portions. This abnormal growth is out of proportion to that of the other parts of the orbit. The superior growth is most marked showing an increased frontal sinus to about double its size.

The possible effects in later life are focal infection and neuralgia of the ophthalmic branch of the fifth nerve.

DELONG, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, March 17, 1921

JAMES B. AYER, M.D., *President, in the Chair*

The Neurological Department of the Boston City Hospital presented these papers:

REPORT OF A CASE OF CERVICAL SYRINGOMYELIA. DR. J. J. THOMAS.

A Pole, 46 years of age, who could speak little English, gave a vague history of paralysis of one side eight years before, from which he recovered. Since that time he had had headache and dizziness, which had become more constant the past seven months, with occasional vomiting, and some trouble with vision. On entrance into the Boston City Hospital he complained of headache and pains in the neck, arms and shoulders, dizziness and staggering as if drunk, and constant tinnitus in the left ear. Examination revealed slight tremor and considerable ataxia of both arms and legs, more marked on the left, and unsteadiness in standing, with a tendency to fall backward and to the left. There was also constant marked nystagmus, which was slow when the eyes were turned to the left, and rapid when looking to the right, and the man complained of diplopia on looking to the right and to the left with one eye as well as when both were open, which was probably due to confusion of vision. The pupils and fundi were normal. The reflexes in the arms were lively and equal. The abdominal reflexes were slightly diminished on the left, and the knee and ankle reflexes were increased and greater on the left. At times ankle clonus was obtained on both sides, and at others only on the left. For a short time Babinski's sign was present on the left, this and the ankle clonus disappearing again. There was apparently hypalgesia and hypesthesia on the lower right side of the face, and the man insisted that he felt nothing on the body below the clavicles. Both labyrinths were normal for the caloric tests, and hearing was normal with tests by tuning forks, except that there was some diminution for both high and low tones on the left, and bone conduction was slightly lowered for low tones on the right. The tympanic membrane and conduction apparatus were apparently normal in both ears. There was slowness of rotation of both forearms. The Wassermann test on both blood and spinal fluid was negative. The spinal fluid was under slightly increased pressure, globulin was increased, later normal, the cells varied in number on different occasions from 5 to 10 per c.mm. The colloidal gold test was negative. Roentgen-ray examination of the head and cervical spine was negative.

An operation was advised, and a subtentorial decompression was done. There was well marked increase of the pressure, but no growth was found in the cerebellum or on the auditory nerve. At the close of the operation the pulse rose rapidly, probably from the effect on the vagus nerve, and the patient died soon after removal from the operating table.

Necropsy revealed nothing remarkable in the internal organs. In the brain the frontal lobes were slightly adherent to each other, and the occipital lobes somewhat flattened, the left more. Section showed no growth in the brain or cerebellum. The upper part of the cord was thin and flattened, and on section showed a cavity centrally located. This cavity at its greatest diameter, which was in the upper cervical region and transverse in direction, was 8 mm., gradually diminishing upward to end in the medulla, and extended below into the upper dorsal region, the length of the cavity being 18 cm. Microscopically the cavity showed the usual aspects of syringomyelia. The cavity in the lower cervical regions was centrally located, and extended back dorsad in the median line nearly to the dorsal surface of the cord, with little lateral involvement. Higher in the cervical region the cavity extended laterally with some involvement of the column of Clark. Still higher there was splitting of the cavity into two, and at other points as many as five, cavities, some always central, but at places extending into the dorsal horns of the cord. The cavities showed in places ependymal cells at the margin. The symptoms, though uncommon in syringomyelia, were probably to be explained by involvement of the restiform body in the medulla, or the column of Clark.

DISCUSSION

Dr. C. M. Campbell thought that the possibility of some associated condition with the syringomyelia ought to be kept in mind, and mentioned the case of a woman in her fortieth year who had been epileptic since 16. She had been taking large doses of bromid and was alcoholic. Since adolescence she had been having frequent convulsions. The sensory tests showed no anomalies of sensation. The patient did not complain of headache, and there was no choked disk. She became increasingly duller. Finally choked disk appeared, and the patient died. At necropsy, in addition to definite syringomyelia, a glioma was found in the posterior part of the brain.

Dr. H. I. Gosline mentioned a case which showed in serial sections the splitting of the cavity as it went upward, and he thought that if all these patients had lived long enough the same peculiarity might have been found. Some observers of syringomyelia have believed that in certain cases other causes than glioma have played a part. In Dr. Thomas' case there was at least a borderline cell count and globulin increase and perhaps some transitory symptoms that might be due to edema. The theory of infection in these cases is not as well supported as the tumor theory. It would be interesting to know whether there was any cellular infiltration in this case of Dr. Thomas.

Dr. J. J. Thomas, in closing, stated that the roentgen ray had not been tried, and that he should not expect it to accomplish much except in cases having diffuse gliosis with formation of cavities. The case reported showed no infiltration about the vessels. From the sections one would probably classify it as a case of abnormal closure of the central canal. The cavity running back from the central canal toward the dorsal septum of the cord with dense neuroglia about it and proliferation of new blood vessels in the tissue shows that it was distinctly a congenital failure of closure. In regard to the existence of two cavities higher up, it is not uncommon to see a great many branches in such a condition, and the distance they run is variable, particularly in the upper medulla. The sections did not show any massing of gliomatous tissue at any one place, simply a thickened layer about the cavities. They showed nothing like a true glioma or even diffuse gliosis.

TYPES OF EPIDEMIC ENCEPHALITIS. Dr. A. W. Fairbanks.

No arbitrary classification can be made. The cases vary widely in the different types. The original conception of the affection is entirely changed in the light of a survey of subsequent clinical experience. Cases seen up to the present time may be grouped as follows:

1. The simple type, characterized by somnolence and lethargy, usually commencing with subjective disturbances of vision, such as blurring of vision and diplopia, but not characterized by definite objective paralysis of the cranial nerves.

2. The common type, in which somnolence and lethargy are associated with definite objective cranial nerve paralysis (our original conception of the disease).

3. The type that commences with general malaise and headache, in which somnolence and lethargy are either absent or appear as late phenomena, but in which cranial nerve palsies may be conspicuous. This type is attended by a feeling of marked muscular weakness, usually forcing the victim to bed, but no paralysis is found on examination. Weakness probably is due to interference with conduction through the motor tracts (pyramids) or more frequently perhaps to interference with conduction through the tracts for muscle-sense in the interolivary region of the medulla. In these cases well marked ataxia in the limbs is often to be found.

4. The bulbospinal type (including those sometimes termed the radicular type). Neuralgic pain is the dominant symptom and usually ushers in the disease. Its character, although usually limited, is strongly suggestive of root irritation. For instance, a case may begin with complaint of intense pain in one hand, later extending up the arm, across the shoulders and down the other arm, thence perhaps down the body; or it may remain unilateral. These patients are likely to develop delirium, often within a few hours of the appearance of the pain, or perhaps some days later. Cervical rigidity is often present, and not infrequently considerable spasticity in the limbs. Somnolence may be absent, or it may appear late in the course of the affection; and the same is true of cranial nerve involvement. Delirium, restlessness and pain dominate the picture.

5. The spastic type without pain. Asthenia is often a conspicuous feature of these cases, and stupor is usually very evident in the early period. Onset may be sudden. Somnolence may be marked for many weeks but often only in the earlier period. Flexibilitas cerea may occur in these cases. Cranial nerve palsy may occur, but as a rule is not prolonged or conspicuous.

6. Myoclonic type. Rhythmical or irregular muscular twitching, more or less general, including the trunk and abdominal muscles. An interesting and singular form in this type is the respiratory form, in which the respiratory disturbance dominates all other symptoms. In these patients the respiration may show a rhythm synchronous with the twitchings, as in an instance cited by the reader, in which for ten weeks extremely rapid respiration persisted.

7. Paralysis agitans type. Onset usually occurs with vague feeling of malaise and general weakness, and often an indescribable sense of discomfort in the legs, soon followed by coarse tremor of the paralysis agitans type.

8. Abortive type. Malaise, headache and often fleeting pains in various parts of the body may appear suddenly and be accompanied by blurring of vision and diplopia. Cranial nerve palsy may appear within a few hours and disappear almost as suddenly as it appeared. Light somnolence is usually present but brief. The duration may be brief but much weakness may remain.

DISCUSSION

Dr. J. W. Courtney stated that the more cases he saw, the stronger his conviction grew that encephalitis is merely the most striking and dangerous symptom of a general constitutional infection. This is well shown by the recent pathologic work of Dr. Harris of the Worcester State Hospital. All patients examined by her showed uniform changes in the spleen and other organs. Dr. Courtney believed that the life history of the filtrable bacillus which causes so-called encephalitis is analogous to that of *Malaria plasmodium*. He has in a given case seen evidence of recurrent activity of the organism at intervals of several months.

Dr. Harold E. Foster, who has been quantitating the sugar content of spinal fluid, reported particularly on a series of cases in which a definite diagnosis of encephalitis was made. In all of the cases except one a definite increase from the normal was noted. In some this increase was nearly twice the normal average and in one fatal case was, eleven hours antemortem, over double the usual reading, being equivalent to a high normal blood sugar. Another case showed a marked rise during an exacerbation of the clinical picture. The blood sugar was normal in all of the correlations made. The ages ranged from youth to old age. Determinations were made from the second day of the disease up to about six weeks. The blood and spinal fluid Wassermann reactions were all negative. There was nothing constant in the cell count, total protein content or gold solution curve. A study of the series would indicate that the increase in spinal fluid sugar is distinctive and in direct relation to the severity of the clinical symptoms but has no relation to the age of the patient or the duration of the disease.

Dr. A. W. Fairbanks, in closing, said that necropsies had been performed on myoclonic cases but no distinctive changes had been found. Almost all spinal fluids showed some increase in lymphocytes, from 5 to 106. The globulin had been practically always increased during the active stage of the disease. The pressure was sometimes increased, but more often not. Approximately seventy-five or eighty cases had been seen. One case in particular showed very well the bulbar syndrome. The asthenia was marked from the start; there was no paralysis but there was marked ataxia in all four extremities. There was no trace of pyramidal trouble. Dr. Fairbanks differed with Dr. Courtney as to the constitutional theory of the disease. He thought the nerve feature the dominant one.

ANHEDONIA; A SYNDROME. Dr. A. Myerson.

Years ago Ribot described the case of a young woman suffering from hepatic disease in which the main mental symptom was the disappearance of satisfaction or happiness. This symptom he called anhedonia. William James in his book, "The Varieties of Religious Experience," declares it to be a passive melancholy in that while there is no dejection or depression, there is loss of zest for living.

For two years a study has been made of psychiatric and neurologic patients from the suggestion thus given by Ribot and James, and the conception of a syndrome has been formed which may be called anhedonia, but which is more inclusive than that of the distinguished Frenchman and American.

Anhedonia is not a disease; it is a syndrome and is made up of four principal changes: First, there is a disappearance of or interference with the great organic sensations—loss of appetite, in many cases loss of thirst. There is almost invariably insomnia, and generally inability to rest well, so that the

patient becomes restless. Usually the patient complains of feeling tired, but when closely questioned this fatigue is not the ordinary normal fatigue; it is a painful sense of incapacity, lethargy or "nervelessness." This last arises, so it seems, from a dropping out of that fundamental organic sensation, the impulse to activity which is one of the primitive organic kinesthetic sensations.

Second, there is loss of hunger, thirst, etc. There is loss of desire for activity in any form, although the patient is restless. This restlessness never takes the form of organized activity. Sometimes, because there is desire for pleasure and the sensations hitherto so pleasant, the individual becomes a pleasure seeker without definite desire except to fill in the painful gap in his mental life. The loss of desire and the lack of the feeling of energy cause ambition to disappear or to be impaired. This dropping out of ambition is exceedingly painful to the patient and deepens the feeling that something has happened to him.

Third, there is loss of the feeling of satisfaction which is the central symptom of anhedonia, as described by Ribot. The patients I have in mind are not definitely depressed; they are not self-accusatory; they do not weep or wring their hands or moan. They complain bitterly that nothing gives them pleasure. Food has lost its savor, drink gives no pleasure, sexual relationship gives but the veriest shadow of the former satisfaction; nothing seems worth while, though it is realized that it is not the fault of things but the fault of the individual's capacity for satisfaction. This is not the dissatisfaction of persons whose desires are not gratified. In such persons desires, strivings and wishes are keen, but in the anhedonic patient there is neither desire nor satisfaction.

Fourth, a curious accompaniment of the anhedonic syndrome which, of course, is not at all limited to it but quite common in mental disease, is the increased diffusion of excitement. Long ago Lotze pointed out that the sick or deficient organism reacts as a whole to excitement much as a child does, whereas the adult, strongly developed organism limits excitement, makes it local. Beard lays great stress on this phenomenon, without so far as I can see, knowing that Lotze had preceded him. Such persons start at a noise; they become confused when many impressions are received at once since every impression produces excitement. They are confused and excited when they meet people in groups, etc., and this excitement is painful because they become self-conscious and develop a feeling of inferiority. In some cases the patient becomes seclusive, not because he is unsocial by nature or because he hates, but because the excitement engendered by meeting people is painful, and his reaction is to avoid pain and the feeling of inferiority. The emotions are readily stirred in a disagreeable direction, and to many of these patients a sense of unreality develops, which is very painful. Doubt and obsession arise out of the feeling of unreality.

The anhedonic syndrome is common, is met after severe illness, often gradually developing long after the illness has passed. It is met in persons in whom some central ambition or desire has been so completely balked as to make achievement or the growth of personal dignity impossible. It is common in the returned soldier. He complains bitterly of loss of appetite, sleeplessness, loss of energy, loss of pleasure, loss of desire and increased excitability under the circumstances named. The most conclusive case seen was in an early stage of dementia praecox. It is often seen as a cyclic disease. Without ordinary depression, without retardation, there is simply a periodic loss of ambition, desire, satisfaction and the loss of the great organic sensations of sleep, appetite, sex desire, etc.

The etiology, it is believed, lies in changes of some sort, produced in the springs of desire and satisfaction; to wit, in the vegetative nervous system, the endocrine glands and the viscera. The study of desire and the attendant phenomena here described is believed to be fully as important to psychiatry as the study of intelligence and emotions. For whether one subscribes to hedonism or not, it is certain that the human being seeks satisfaction for his desires, and that desire is largely based on instinctive mechanisms of a prime order which constitute the foundation of human personality.

DISCUSSION

Dr. J. W. Courtney remarked that an observation like Dr. Myerson's is a signal advance in the comprehension of morbid mental states. The sensory and sensorial disorders which underlie the anhedonic state are not measurable by the esthesiometer or any other such crude instrument. They nevertheless denote an adynamic condition so marked in intensity that the perception and diffusion of stimuli necessary for apperception cease. When the anhedonism reaches a certain degree of profundity the feeling of reality is lost, and it is only by constant argumentation that the victim of this feeling succeeds in convincing himself that it is *his* hand with which he performs certain movements, *his* feet which carry him about, *his* voice with which he speaks. In a still more advanced state of depression the patient fails to convince himself by argument, and harbors the delusion that the various parts of his body are not real living parts, that he is nonexistent; and he finally carries his notions of nihilism to the point where they embrace all the phenomena of the universe.

OBSERVATIONS ON THE TREATMENT OF PARESIS. Dr. LeRoy A. Luce.

Arsphenamin and arsphenamized serum have been used at the Boston City Hospital in the treatment of about 500 patients with neurosyphilis during the past nine years. Two thousand intradural serum injections were given by either the Swift-Ellis or the Ogilvie method; these being the favored methods. No severe reactions or deaths followed. Dr. Luce presented two cases of paresis that had received a considerable amount of treatment extending over a long period of time.

Case A: A man, 42 years of age, gave a history of chancre fifteen years before symptoms of nerve involvement began to appear. Hallucinations of sight and hearing and mental deterioration predominated. The blood and spinal fluid were positive, globulin positive, cell count 46, and the colloidal gold test showed a paretic curve. This patient received seventy Swift-Ellis serum treatments extending over eight years. The blood and spinal fluid remained Wassermann-fast throughout the whole period of treatment, and the colloidal gold test did not change from the paretic curve. The cell count changed from 46 to 3.

Case B: A man, 35 years of age, with a history of chancre five years before symptoms of nerve involvement, had not received proper and efficient antisyphilitic treatment during the primary and secondary stages of the disease. He had delusions of the expansive type and was mentally exalted and erotic. The blood and spinal fluid were positive, and the colloidal gold test showed a paretic curve. This patient had received seventy-five treatments in seven years. The blood and spinal fluid remained Wassermann-fast, globulin positive, and the colloidal gold test did not change from the paretic curve.

These patients with undoubted paresis received about the same number of treatments. One man was able to do simple work, while the other was helpless both physically and mentally. These observations were reported to bring up a discussion on the value of treatment in paresis.

DISCUSSION

Dr. A. W. Fairbanks expressed the opinion that from a clinical point of view to treat a definite case of paresis was a waste of good material and more valuable time.

Dr. A. H. Ruggles mentioned three patients who had left the hospital in the past year, all of whom were capable of working, one now with negative findings and two with positive findings. On the other hand, he had observed two patients, neither of whom had had any treatment, one of seventeen years' duration and one of twenty-one years, both of whom are still alive.

Dr. F. H. Packard stated that the results in treating general paralytics at the McLean Hospital had not been encouraging; indeed, treated patients had done worse and gone down hill faster than most of the untreated ones. None of the treated patients had had remissions while a considerable percentage of the untreated patients had not only held up for considerable periods, but some of them had had strikingly good remissions of considerable duration—remissions so good as to make optimistic relatives feel that they had recovered.

Dr. H. C. Solomon stated that an analysis of the figures of state hospitals in Massachusetts show that up to 1916 about 2.5 per cent. of the patients with general paresis who were untreated had remissions, none of which lasted one year, and of the treated patients 25 per cent. had remissions some lasting for four, five or seven years. These figures indicate the value of treatment of patients whose cases had been diagnosed as general paresis.

REPORT OF A FEW CASES OF CRANIAL NERVE PARALYSIS OCCURRING DURING AN EPIDEMIC OF ACUTE POLIOMYELITIS.

Drs. Robert H. Courtney and William E. Savage.

Of twenty-four patients with acute poliomyelitis who were admitted to the Boston City Hospital during the four months ending Dec. 1, 1920, five showed involvement of the bulbar region alone, one of the bulbar and a segment of the cervical region, and eighteen had purely spinal types of the disease.

Of the five patients with bulbar cases four were males and one a female. The ages in four cases were between 11 and 13 years, and in one was 3½ years. Three of the patients recovered but with permanent (five months after onset) cranial nerve paralysis; two died, one from involvement of the respiratory center of bronchopneumonia.

Two of the patients showed complete paralysis of the lower branches of one facial nerve and weakness of the upper branches. The other three showed involvement of the glossopharyngeal nerve with regurgitation of food through the nose. In these, laryngeal diphtheria was excluded both clinically and bacteriologically. The spinal fluid of all showed an increased cell count varying from 80 to 200 with a slight preponderance of lymphocytes. The treatment of all except one consisted of lumbar puncture repeated at intervals of two days. In one case convalescent serum was given, but improvement had already begun.

Necropsy was obtained in one case. Gross examination of the brain and spinal cord was negative. Microscopically there were extensive perivascular lymphoid cell infiltration and fairly diffuse and focal infiltration of endothelial leukocytes about the small capillaries of the pons and medulla. The pathologist reported a microscopic diagnosis of anterior poliomyelitis.

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Eighty-Eighth Regular Meeting, April 5, 1921

FOSTER KENNEDY, M.D., *President, in the Chair*

A CASE OF POLYCYTHEMIA WITH NERVOUS SYMPTOMS. DR. MOSES KESCHNER.

Dr. Keschner presented a patient suffering from this disease. Polycythemia, or erythremia, is a condition of the blood in which the number of red blood corpuscles is increased above 5,000,000 per cubic millimeter. It may occur as a result of physiologic stimuli or in disease as a result of pathologic stimuli. It may be relative, that is, without real overproduction of red cells. In absolute or true polycythemia the total number of red cells in the body is actually increased. This is a disease of unknown origin characterized by persistent erythremia due, it is thought, to excessive erythroblastic activity of the bone marrow, or to a failure of destruction of the red blood corpuscles. Some also ascribe it to faulty internal secretions, especially of the suprarenals. The nervous manifestations have hitherto received little attention, in contrast to those of the anemias. The symptoms are: headache, dizziness, visual disturbances, paralysis (motor and sensory), pain, muscular twitchings, convulsions, speech disorders, mental, vasomotor and trophic disturbances. There is general or localized pain, a vague aching and feeling of fulness and throbbing over the entire body, especially in the head. Some of the patients show defects of memory and general symptoms of neurasthenia; they are emotionally unstable and have a tendency to become easily depressed or excited and at times are delirious. Insomnia is a common symptom. In the vasomotor sphere, hyperhidrosis is frequently found.

Eye symptoms are common and frequently the first noted. Some patients complain of easily induced fatigue from the use of the eyes. Palsies of the external ocular muscles are quite common. These may give rise to diplopia. Scintillating scotomas, transient blindness and hemianopsia are not infrequent. Ascher has described in this disease a form of asthenopia without refractive errors, in which the patients give a history of repeated but unsuccessful efforts to get proper adjustments of glasses. The ophthalmoscope reveals changes in the fundi which are characteristic of the disease. The veins are distended and engorged so that they appear much larger and darker than the arteries; they are tortuous and may show varicosities. The disk may be normal, or slightly reddened, or it may have the appearance of mild optic neuritis.

The nervous symptoms in the anemias are due to degeneration of the neurons brought about by toxemia. The pathologic bases of the nervous symptoms in polycythemia are vascular. They may be thrombotic or hemorrhagic, and cause softening which is not due to arteriosclerosis.

The patient, a 30 year old Russian tailor, was admitted to the Mount Sinai Hospital complaining of headache and salivation from which he had been suffering for the last eight years. The salivation, as well as the headache, was almost constant. His symptoms had been attacks of tonsillitis in the past, headache, salivation, dizziness, vertigo, blurred vision, anorexia, hemorrhoids, congestion of face and eyes, insomnia, uncontrollable hemorrhage after tonsillectomy, flushes of heat and cold, tinnitus and epileptiform seizures. Blood examination revealed: hemoglobin, 170 per cent. (Kuttner); 9,800,000 red blood corpuscles; 340,000 blood platelets, and 8,000 white blood corpuscles. The

red cells were microcytes and showed some slight central pallor. A later examination revealed: hemoglobin, 170 per cent. (Kuttner); 11,456,000 red blood cells; 410,000 blood platelets, and 7,600 white blood corpuscles. The color of the blood stream in the veins of both disks was reported to be dark purple, much darker than normal. The arteries were normal. The visual fields were normal. The patient was treated with atropin until there was marked dryness of the throat and dilatation of the pupil, but the salivation did not diminish, nor did local astringents applied to the mouth have any effect on it. After the withdrawal of 10 ounces of blood from the median basilic vein, the patient stated that his headache and dizziness were slightly relieved.

DISCUSSION

Dr. B. Sachs asked concerning the patient's subjective state. Dr. Keschner resummarized the chief symptoms. The patient, when asked if he felt dizzy or sleepy, replied in the affirmative. Dr. Sachs asked how the disease could be recognized.

Dr. Keschner said that it was recognized in this patient by salivation, headache, attacks of dizziness and epileptiform seizures. It was first suspected when great difficulty was experienced in drawing blood from the vein in trying the Wassermann test. There was no evidence of other nervous disturbances. The appearance of the veins in the disks and the headache were the main factors. Dr. Keschner's theory was that the salivation was central. It was not affected by atropin. The saliva had not been examined.

Dr. C. L. Dana said that a diagnosis of polycythemia could not be made from any nervous symptom.

Dr. J. H. Leiner said that in the last six months he had seen two cases of this disease in which there were no nervous manifestations at all. An increase in blood cells was present. The throat was curiously stippled like the throat in scarlet fever. There were present a black pigmentation along the mucosa, a characteristic congested eye and a cyanotic flush of the face. A woman, 55 years of age, who had polycythemia vera, had high blood pressure. Some of the skin conditions he thought were endocrine in origin.

Dr. Rosenthal, by invitation, said that a different type of polycythemia was present with the high blood pressure. It was described a long time ago by Geissler and was called polycythemia hypertonica. Here the blood pressure and pigmentation were not so marked. In these cases tuberculosis must always be ruled out, since polycythemia might be secondary to it.

Dr. Keschner said that the disease had been treated by roentgen-ray exposure of the spleen and of all of the long bones, and had been accompanied by improvement. Some writers also report encouraging results from this treatment; others say that some of these patients get better without treatment, and that the improvement simply represents a spontaneous remission. The injection of epinephrin causes an increase in the red blood cells. The condition is by some thought to be due to an adrenalemia.

COMBINED NEUROLOGIC PERCUSSION HAMMER. Dr. J. H. Leiner.

Dr. Leiner demonstrated a percussion hammer half the size of the ordinary hammer when closed, a little shorter when open. The design is that of the Roman battle axe; the balance is delicately adjusted, and it is weighted by metal and a strip of soft cushion rubber to prevent trauma. The back had originally been made of a soft piece of rubber. It is now replaced by a hard piece set in by a set screw. The original hammer also turned from side to

side and swerved. In this model, therefore, a groove has been cut in the bottom of the shank with a pin running through it. At the other end of the hammer there are three tubes to contain the instruments commonly used. These are a needle (very sharp) which can be pushed out by a little button, a colored orange stick to test color vision, which snaps back when not in use, and a camel's hair brush for touch. This hammer obviates the necessity of always carrying pins with one. It can be used for making the Babinski test, also as a tongue depressor. It makes absorbent cotton unnecessary. These hammers are made by N. A. Low and Co., New York, and cost $7.50 each.

DISCUSSION

DR. SACHS said that he had been using this instrument for the past weeks and found it convenient.

A CONTRIBUTION TO THE HISTOPATHOLOGY OF PORENCEPHALUS. DR. J. H. GLOBUS.

This paper will appear in a future issue.

DISCUSSION

DR. B. SACHS felt that this work corroborated a suspicion which he had expressed about thirty years before. It still remains a question whether all cases can be explained by such defects. The occlusion of one of the branches of the larger arteries is not unknown. He asked whether Dr. Globus had sufficient evidence to show that the porencephalic defect occurred during the prenatal period or whether it could have been developed after birth.

DR. FOSTER KENNEDY said that only a few days ago he had seen a child with a definite nuclear aplasia of the right seventh and hypoplasia of the right sixth nerves. The mother had been desperately ill with influenza at six months' pregnancy. At the time he had thought that the failure to develop was due to encephalitis in the fetus.

DR. GLOBUS, in answer to Dr. Sach's question, again stated that there was absolutely no history of illness in the postnatal life of the child. The mother had had uterine inflammatory condition early in pregnancy and before conception. Lesions both chronic and subacute had been found which therefore must have taken a long period to develop. The disease must be placed in the later part of intra-uterine life or in early childhood.

SOME FEATURES OF THE NEURO-PSYCHIATRIC WORK ON ELLIS ISLAND. DR. B. ONUF.

Dr. Onuf said that this was the first time that the question of immigration had been brought up before the Society. The aim of Ellis Island was to control admissions to the United States, to exclude undesirables and to restrict the entry of those who might become public charges. The question of undesirability, from the medical side, involved personal shortcomings and disabilities, transferable diseases and hereditary factors. Laws provide for the absolute exclusion, by a Board of Special Inquiry, of idiots, imbeciles, feeble-minded, epileptic and insane, constitutional psychopathic inferiors and chronic alcoholic patients. The mentality of physical defectives required certification. The law of exclusion held without appeal for all classes of defectives except the physical, on which a board of special inquiry passes. The individual may appeal from the decision of this board, in which case the question is passed on to the Secretary of Labor who may grant admission under bond.

The medical work consists in recognizing the undesirables. The general procedure is as follows: The immigrants pass before the examiners and medical inspectors in line. At the head of the line stands a physician examining the eyes to make sure that no cases of trachoma pass. From the survey of immigrants as they pass, a rough general examination is completed, and persons presenting any abnormality are marked with chalk, the letters to designate the part affected, such as eyes, lameness, hernia, etc. The patient marked is then further examined. The one marked "L" for lame, for example, may show spastic paraplegia or locomotor ataxia, or hemiplegia, or progressive muscular dystrophy or multiple neuritis, etc. The examiner is called on to differentiate between countless causes. It is also a usual practice in making the routine examination to speak a word or so to each person and thus elicit a reply and in this way investigate speech. A total examination gives further information in regard to muscular atrophies, etc. It is then the duty of the medical officer to certify the neuropsychiatric conditions found. Sometimes the examiners differ. In cases of doubtful diagnosis the person is usually certified as having organic disease of the central nervous system.

The difficulty of arriving at a diagnosis is great. The immigrant often will not give any information, or if he does, it is intended to delude. The same difficulty is encountered in examining mental defectives and psychotic patients. Here the Binet-Simon test cannot be relied on because of the variety of standards and environment represented by these aliens. For instance, some immigrants have never seen electric light, they have no knowledge of dates, the number of months in the year, the days of the week, etc. The conditions imposed by foreign languages is also exceedingly difficult. Interpreters miss the points of interest and value to physicians. In a psychotic case, for example, the physician wants to know just what the patient says. The interpreter tries to make sense out of it and thus slurs the abnormalities in the current of thought.

Where a conclusion cannot be reached immediately the alien is sent to the hospital for observation. Thirty years ago there were no observation facilities. Now there is complete equipment and laboratories. In many cases, such as trachoma, mental deficiencies due to hypothyroidism or athyroidism and other conditions arising from disturbances of internal secretion, a diagnosis is not made until the result of treatment is seen. In other cases the diagnosis is not made until confirmed by bacteriologic examinations, as in leprosy, favus, ringworm, etc. Weekly conferences are held at which all interesting cases are presented and discussed and outside consultants come to lecture, etc.

DISCUSSION

Dr. H. V. Wildman, Jr., said that with the great influx of peoples from southern and eastern Europe the problem of keeping out those with mental defect was becoming one of the greatest importance. The detection of feeble-minded immigrants is a separate and distinct specialty. Suggestions have been made that the Army B test be applied to arriving aliens, but experimental work with group tests in general and the B tests in particular have not given good results. The largest number of mentally defective aliens have been detected by a short personal interview with the individual alien during the line inspection.

A discouraging feature of the work at present is that many persons mandatorially excludable, are landed for various periods of time at the discretion of the Secretary of Labor. Lack of proper supervision allows some of these persons, through various contingencies to land permanently in the country.

Dr. I. J. Sands said that many of the immigrants who became ill might later be found in Bellevue. Many of those who were admitted to the observation wards echoed many of the horrors that had been occasioned by the late war. Thus, a Russian developed a psychosis while on the steamer on getting white bread to eat; he had been living on straw for several months, and the sight of real bread caused him to react in a manic-depressive fashion. Another case was that of a Russian Jewish girl who had thrown her brother's children from a window. She was in a state of depression when she came to this country, and after six months a manic state developed. Her original depression was undoubtedly occasioned by the hardships encountered abroad and by the difficulties met with while traveling through Russia and Poland. Dr. Sands thought that those immigrants that might later suffer from a neuropsychiatric condition to the extent of making them public charges, could hardly be detected by means of the ordinary so-called line examination. Cases of multiple sclerosis, paresis, etc., would hardly reach the boat; while cases of high grade mental deficiency, manic depressive phychosis in the mild and early stages and simple praecoxes, might easily escape detection. With an increase in the number of examining physicians, and the assignment of those men who have a knowledge of several of the foreign languages to Ellis Island, the number of undesirable aliens admitted would be materially reduced.

Dr. H. V. Wildman said that the percentage of mental defects picked up at Ellis Island was three times that of the feebleminded in institutions of the state of New York. Certificates of mental defects are issued only in cases about which there is no doubt.

In regard to the psychoses, the country is protected by the law which provides for deportation of aliens who become insane within five years of landing. A similar provision for cases of mental defects detected within five years after landing would seem an additional and desirable safeguard if it could be incorporated in the next immigration law.

Dr. Onuf, in closing the discussion, admitted that the examination could be made more thorough if the examining board had more men. The force is sometimes inadequate. When immigration is heavy fewer certifications are issued since the examiners have not time enough. Yet in spite of the rush a good number of psychoses are certified.

Book Reviews

LEITFADEN DER ELECTRODIAGNOSTIK UND ELEKTROTHERAPIE FUER PRAKTIKER UND STUDIERENDE. By Prof. Dr. Toby Cohn, Neurologist in Berlin. Sixth Edition. Price 30 M. Pp. 226. Berlin: S. Karger, Karlstrasse 15, 1920.

The first edition of this work was published in 1898. It is apparent that the author has kept in close touch with all advances along physiologic, experimental, diagnostic and therapeutic lines. The book differs from the conventional presentations of this subject in being readable and at the same time sufficiently complete to serve as a reference work. It is divided into two parts; Part I takes up electrodiagnosis, Part II electrotherapy. There are numerous footnotes and insertions that are particularly interesting and instructive.

The physical characteristics of the electric current and the various types which find application in medicine are discussed in Chapter I. The second chapter deals with the physiologic problems that are involved. The methods of examination are in a general way sufficiently understood and need no further comment; a number of valuable technical hints, however, are given to overcome the difficulties that are ordinarily left for the tyro to discover and contend with.

In the discussion of various types of degeneration and changes in reaction that are found in muscles and nerves, the book conveys an assurance that the writer has had vast experience along these lines, since he also confesses to having difficulties in interpretation and departs from the tiresome orthodoxy that results in so much discouragement when one attempts to put what one reads into practice. In spite of this, unity and coherence are retained throughout in a most satisfactory way.

The electrical examination of sensory organs, such as the eye, the cochlear and the vestibular apparatus, is discussed but is not stressed as being very practical. This also applies to the discussion on the examination of cutaneous sensibility. The presentation of resistances is enlightening in that a subject is taken up that is usually neglected.

Part II is devoted to electrotherapy and dispels any lurking preconception concerning the writer's over enthusiasm. While he takes up the various methods that are employed in electrotherapeutics, he is always cognizant of the psychic factors. The physical action of the current on which much of the benefit from treatment depends is based on wandering of ions, thermic reactions, chemical reactions, ionphoresis, and some bactericidal properties; the physiologic action is dependent on vasomotor effects, changes in electrotonus, which is local rather than general and of limited value, contraction of the smooth and striated muscles, cutaneous reactions, and secondarily, effects on blood pressure and metabolism. Treatment of various diseases of the peripheral nerves, muscles, spinal cord and brain is taken up somewhat in detail. The treatment of the neuroses is considered rather briefly, probably because results depend almost entirely on psychotherapeutic factors of which electrotherapy should be only a small part. On the whole, the treatment of internal organs by such methods as necessitate the insertion of various electrodes is con-

demned, since the benefit that is derived is often questionable and the harm that may be done, considerable. On account of the effects on the mucous membrane incident to polarization, the use of the galvanic current is discouraged altogether.

Various types of apparatus, their construction and different advantages are dealt with. The chapter on franklinization is brief and quite in line with the discarding of the imposing influence machines from the modern office equipment, since no particular value is found here that cannot be obtained from other methods of obtaining the current. Arsonvalization, introduced by Tesla, is regarded with skepticism. The sinusoidal current is strongly condemned, since deaths have followed its application. Treatment with the Leduce current and with electromagnetism is briefly explained and discussed.

In the last chapter the condensor apparatus of Zanietowski is described. While comparatively little is known of it, the writer believes that examination by this method will become increasingly important, since it permits an accurate measurement, in microfarads, of the amount of electricity discharged. It has the advantages of being little influenced by cutaneous resistances and having a constant value as compared with those of the ordinary galvanic current as shown in Stinsing's tables.

THE BASIS OF PSYCHIATRY (PSYCHOBIOLOGICAL MEDICINE). A Guide to the Study of Mental Disorders for Students and Practitioners. By ALBERT C. BUCKLEY, M.D., Associate Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. Cloth. Price, $7. Pp. 447, with 79 illustrations. Philadelphia: J. B. Lippincott Company, 1920.

Here we have a book of major length written from one courageously held point of view. Recoverable psychoses are regarded by the author as "reflections of some bodily disorder which through its effect on the organ of adjustment prevents the patient from making appropriate adaptation to environmental conditions." It follows that interest must be centered on those essential "bodily disorders" and "the organ of adjustment."

So we have a long and remarkable introduction in the first seven chapters. Attention is characteristically called to things outside of the skull, to the field of internal medicine, to muscles and glands which are meeting, or falling short of, or exceeding the demands made on them by a changing environment. A description of the development of the nervous system leads from Gaskill and Parker to Sherrington. In discussing salivary and gastric secretion, the author's goal is shown by his quotation from Pawlow: psychic activity "is nothing but an endless chain of reflexes." The conditioned reflex and the autonomic nervous system are again given the honor of providing the bridges to lead from laboratory to clinical observations.

Central localization and the anatomy of the brain in general are carefully written and proportioned to the needs of the reader of a textbook on psychiatry. A section on heredity is well illustrated and diagramed.

Mental development and psychologic processes are given nearly fifty pages, with at first comparisons with animal development and later much reference to the visceral accompaniments of mental states. The "unconscious" is discussed in the last four pages and with it freudianism in general.

No more interesting discussion of etiology is needed than that found in chapters 6 and 7, the minimizing of psychic causes making the handling of the problem more striking. Mental patients fall into two great classes:

"1. Those who develop psychoses irrespective of environment; for example, paranoia and manic-depressive psychoses. 2. Those who develop psychoses provided there exist the proper environment and exciting causes; for example, puerperal and other toxic psychoses, general paralysis and organic dementias —*accidental* psychoses." How many mental diseases may, after the fashion of general paralysis, develop years after a "mild" infectious process? The author thinks more in terms of widespread toxins than of focal infections, stressing disturbances of internal secretions, general effects of syphilis and tuberculosis, and in particular citing the importance of alcohol in mental disorders "produced" by head injury.

The chapters on classification, symptomatology and methods of examination are not unusual. Weston has called attention to some errors and omissions in the section on laboratory technic, and a manual other than this general work should naturally guide the laboratory worker.

The different psychoses are treated under nine headings in the second half of the book. All are defined in subheadings as reaction types due to infection, toxin, or degenerative brain process, or to unknown cause. The dementia praecox group appears "during the evolutive period, arising out of an inherent biogenetic defect, terminating frequently in neuronic degeneration, and characterized by an interference with the adaptive mechanism through inefficiency of the sensory, emotional, and volitional processes." The manic-depressive group is considered as a quantitative disturbance of everyday reactions. The psychoneuroses are "generally regarded as of psychogenic origin." The attractive chapter on "Psychoses with Somatic Diseases," is abstracted in the following paragraph as typical of the author's method:

These are mental "reactions dependent on functional disturbances of the organism due to" the inseparable trio "infection, exhaustion or toxemia, affecting the sensory motor and associating mechanisms, characterized by delirium, confusion, and physical prostration." They greatly concern the general practitioner who may meet delirium before, during and after a fever. Mental states are found which vary from insomnia and restlessness with depression through hallucinosis, confusion to wild excitements or long neurasthenias. On the physical side may appear twitching of muscles, nausea, constipation, dry or clammy skin and paresthesias. The pathologic condition consists of engorgement of membranes, disintegration in cortical neurons and cells of basal nuclei or hemorrhagic extravasations into the brain substance. In treatment we try to help an organism which not only cannot cope with the environment, but which is also poisoning itself, and needs a careful physical examination and a nurse trained in general medicine and psychiatry. Liquid diet is given in moderate amounts frequently with much attention to water supply, baths, and administration of alkalies. The minute directions should be carefully followed by any general physician who has to treat these infective-exhaustive conditions—which have a high mortality because in general their treatment is not understood.

In brief, this is a book worth owning, unusual in point of view and broad in scope, not attempting to cover the field of "psychic mechanisms," but emphasizing the broad foundation of psychiatry and many practical and exceedingly good points in treatment.

TRAVAUX NEUROLOGIQUES DE GUERRE (PAPERS ON WAR NEUROLOGY). By GEORGES GUILLAN and J. A. BARRÉ. Paper. Price, 18 francs net. Pp. 460, with illustrations. Paris: Masson et Cie, 1920.

Introduction: This book of 460 pages is made up of a preface by Pierre Marie and six chapters:

Chapter 1, Neurological Symptomatology (fifteen papers); chapter 2, Wounds of the Brain (eight papers); chapter 3, Wounds of the Spinal Cord (seven papers); chapter 4, Concussion Without External Wounds Resulting from the Explosion of Shells (twelve papers); chapter 5, Pathology of the Cranial and Spinal Nerves (eight papers); chapter 6, Varia (five papers).

The first chapter is concerned with reflexes normal and abnormal. The second chapter is more interesting. The papers on a thalamic syndrome, on hemorrhagic meningitis without injury of the dura and on the late results of trephined cases are among the best.

The chapter on spinal cord wounds is full of interesting papers. That dealing with complete transverse lesions showed, among other things, that the deep reflexes were absent (Bastian's law) in thirteen out of fifteen cases. The reaction to plantar stimulation was present in fourteen out of fifteen cases, but was flexor in type. The other case showed no reaction. Other interesting observations on reflexes, both somatic and visceral, are to be found in this paper.

The paper on organic paraplegia from wounds of the vertebrae without dural rupture and that on lesions of the cauda equina contain much valuable material.

The chapter dealing with organic lesions after shell explosions which cause no wounds is too lengthy and detailed (eighty papers) for a review. It takes up the various aspects of this important subject quite thoroughly.

The next chapter contains papers on oculomotor paralysis during paratyphoid, ocular paralysis from picric acid poisoning with demonstrations of picramic acid in the spinal fluid and various unusual types of spinal nerve paralysis.

The last chapter deals with various subjects: spinal fluid hypertension following ligation of the jugular vein, periodic paralysis (Westphal-Goldflam), Landry's paralysis following antityphoid vaccination, radiculitis and gas poisoning.

The book gives a good idea of war accidents involving the central nervous system. The subject of involvement of peripheral nerves is not brought up except in a few case reports. The book is worth reading, is well written and well illustrated, and has a variety of material which should be of interest to both the medical and surgical neurologist.

THE CROONIAN LECTURES ON THE PSYCHOLOGY OF THE SPECIAL SENSES AND THEIR FUNCTIONAL DISORDERS. By ARTHUR F. HURST, M.A., M.D., OXON, F.R.C.P., Physician and Neurologist to Guy's Hospital. Pp. 122. London: Henry Frowde, Hodder & Stoughton, 1920.

After carefully reading this work, one is tempted to say "a little book with a big I." Essentially it is clinical, a report of cases of hysteria (mostly in soldiers) seen by the author. Of psychology, either of the special senses or of hysteria, there is just a trace. What strikes the reader is the constant emphasis placed on the discoveries and opinions of the author. The former

are almost entirely rediscoveries of things known for many years or familiar to all neurologists who have done war work or read of it. The latter are for the most part either tiresomely trite or not those of a mature neurologist.

The psychology, what there is of it, is naive to a degree—quite "childlike and bland." Briefly stated, it is this: The person hysterically blind is so because he does not look, the hysterically deaf is so because he does not listen and the hysterically anesthetic does not feel because he does not pay attention. The mechanism is that the lack of attention and failing to look and listen cause a break in the sensory afferent tract somewhere in the brain stem by retraction of dendrites at a synapse.

But if the book is neurologically and psychologically weak, not to say amateurish, and the accomplishments of the author exalted to an unwarranted degree, it is still a very readable and interesting record. Hysteria, the protean, is always interesting, and if the author does not greatly edify us, his cases do. Some of his own work is also interesting. "Twenty-nine healthy and intelligent individuals, of whom twenty-five were medical students, who had, however, not yet worked in the wards or read any clinical medicine, were asked to pretend that they had been in a railway accident, and that they were attempting to swindle the company by claiming compensation because of paralysis of the right arm and leg, which they alleged had resulted. They were to act as well as they could the part of men trying to persuade the medical examiner that they were really suffering from paralysis." The results were quite comparable to the symptoms of traumatic hysteria.

The therapeutic procedures employed are along the lines of persuasion and suggestion with which all neurologists are familiar, and which are good. But the author falls into the old error of post hoc propter hoc. Because his theory of hysterical deafness when explained to the patient helps to cure him, the author concludes that it is the real explanation of the deafness. When a magnet transfers anesthesia or banishes paralysis we do not conclude that there is virtue in the magnet, but that the patient has accepted a helpful idea. That Hurst's conception of hysterical blindness and deafness may appeal to and be accepted by an intelligent person, is shown by its acceptance by Hurst himself.

As a clinical assistant the book will be very useful to those not familiar with hysteria, and for all it is interesting and stimulating.

A STUDY OF WOMEN DELINQUENTS IN NEW YORK STATE. MABEL RUTH FERNALD, MARY HOLMES STEVENS HAYES and ALMENA DAWLEY, with a statistical chapter by BEARDSLEY RUML, and preface by KATHERINE BEMENT DAVIS. Pp. 542. New York: The Century Company for the Bureau of Social Hygiene.

The purpose of this comprehensive study was to attain a more adequate understanding of women delinquents. The groups of women studied include 102 at Bedford Reformatory; 88 at Auburn State Prison; 76 at the New York Magdalen Home; 110 at the New York County Penitentiary; 109 at the New York City Workhouse and 102 probation cases from the women's night court of Manhattan and the Bronx, making a total of 587 cases. The results are compared with those found by other investigators both in groups of delinquents and of general population. The principal topics treated are: nature and extent of delinquency; age; habits with respect to use of tobacco, alcohol

and drugs; general social factors; nativity and color; early home conditions and family status; educational background, occupational, history and economic efficiency; sex irregularities and mental capacity.

In one of the most valuable chapters in the book mental capacity is correlated with other factors which might influence delinquency. The authors find that the average mental capacity of women delinquents is somewhat lower than that of the average of women in the general population. They do not, however, agree with Goring that constitutional factors are of greater importance than environmental factors as determinants of crime. The fact is emphasized that delinquency in women cannot be traced to any one predominant factor but results from a large variety of interrelated causes.

The study seems to indicate that native born colored women have the highest rate of delinquency, native born white women a considerably lower rate and foreign born white women a still lower rate.

As a whole, this is a valuable contribution to our knowledge of women delinquents and great credit is due the authors for their thorough investigation of individual cases. One could feel surer of the statistical conclusions if the number of cases studied were larger. The statistical tables and drafts are admirably presented, and the limitations of each are clearly set forth. The frequent use of standard deviations and ratios of correlations with the accompanying discussions of the probability of error make some of the discussions too technical for the average reader. This painstaking statistical work, however, increases the value of the book to the student of social science.

GRAPHIC METHODS IN HEART DISEASE. By JOHN HAY, M.D., F.R.C.P., Honorary Physician, Liverpool Royal Infirmary; Honorary Consulting Physician, Ministry of Pensions; Later Consulting Physician in Diseases of the Heart (Western Command); with an Introduction by SIR JAMES MACKENZIE, M.D., F.R.C.P. Pp. 172. London: Henry Frowde, Hodder & Stoughton, 1921.

This book is chiefly concerned with a discussion of the use of the polygraph in disease of the heart. The exposition of the subject is clear, concise and free from unnecessary and bewildering details. The mechanism of cardiac irregularities and the aid to be derived from the polygraph in their interpretation are made plain. The numerous figures, most of them original, are, because of the fine quality of paper employed, of unusual excellence. In brief, we know of no bettter discussion of the essentials of polygraphy than is given by Hay in this small volume.

Since the publication of the first edition twelve years ago, the electrocardiograph has largely supplanted the polygraph. Some even go so far as to say that the polygraph is now of historical interest only. But because of its relative cheapness and its portability, the polygraph still remains the more serviceable instrument for the practitioner. Hay has added a brief sketch of electrocardiography with several typical electrocardiograms that will give the reader a glimpse of what may be revealed by this wonderful machine.

The question is often asked: Of what practical use are the polygraph and the electrocardiograph? The last page of the book contains the best answer to this question we have ever seen. The answer, as given by Hay, is that these instruments "create a new interest." He expounds this text in a few short but illuminating sentences.

TROPICAL OPHTHALMOLOGY. By ROBERT HENRY ELLIOT, M.D., B.S. (LOND.), Sc.D. (EDIN.), F.R.C.S. (ENG.), Lieutenant-Colonel, I. M. S. (Retired), Late Superintendent of the Government Ophthalmic Hospital, Madras and Professor of Ophthalmology, Medical College, Madras; Honorary Fellow and Gold Medallist of the American Academy of Ophthalmology and Otolaryngology; Lecturer in Ophthalmology, London School of Tropical Medicine; Ophthalmic Surgeon to the Seamen's Hospital Society, and to the Hospital for Tropical Diseases, Endsleigh Gardens, London. Pp. 502, with 7 plates and 117 illustrations. London: Henry Frowde, Oxford University Press, Hodder and Stoughton, Warwick Square, E. C., 1920.

This work is practical and readable, the fruit of about twenty years of experience in India. The author shows that tropical ophthalmology has become a special study, as much from the habits of mind and body of the natives as from the influences of a tropical climate. For example, we are confronted with a prophylactic operation for glaucoma. Besides a comprehensive discussion of the various tropical parasitic diseases of the eye, the author gives careful attention to the more unusual external diseases, such as rare forms of keratitis, Herbert's corneal plaques, conjunctival granuloma, and other conditions. An interesting and instructive account is given of the native operations of couching for cataract, in which one makes a capsulotomy with a needle knife before making the corneal section. The very short chapter on ophthalmoscopic appearances in the tropics (by Lieut.-Col. H. Kirkpatrick) seems all too short, and it is to be hoped that a subsequent edition may devote more attention to this branch. The author's discussion of eye conditions in general diseases in collaboration with Professor Miyashita of Japan is very full. The book is generously illustrated with photographs, original drawings and excellent colored plates.

UEBER DIE JUVENILE PARALYSE. TONI SCHMIDT-KRAEPELIN, Berlin, Julius Springer. Pp. 124. 1920.

In this monograph is presented the first part of a consideration of congenital syphilis in its relations to mental abnormality, which is finally to include the neurologic disturbances of idiocy and imbecility, epilepsy, syphilis of the brain, and even psychoneurotic tendencies in the apparently sound offspring of syphilitic patients.

Dr. Kraepelin presents only juvenile general paralysis, in thirty-nine cases of pure type, seven doubtful cases, and seven cases in which serologic, but not clinical, positive findings (pre-paralysis?) were obtained. She makes a thorough analysis of histories, symptoms and events in the course of the disease processes. Thirty-seven tables explain this part of the text alone, including tables showing metasyphilis in parents, Wassermann reactions in relatives, the preliminary symptoms, neurologic signs and treatment. A section follows which gives the individual records in painstaking detail with photographs in full length of the pure types. Consideration of the doubtful cases is clarified by more tables (up to Table 53), including a comparison of constitutional traits, stigmas, neurologic, physical and mental symptoms.

The conclusions are in general not unexpected. When one adds the frequency of focal symptoms, ataxias, pseudo-choreas, and infantilism to a known hereditary cause, it becomes more than ever necessary to rule out general paralysis when diagnosing the neurologic diseases of children.

KURSUS 'DER PSYCHOTHERAPIE UND DES HYPNOTISMUS (A COURSE OF PSYCHOTHERAPY AND HYPNOTISM). Von San.-Rat. Dr. Georg Flatau, Nervenarzt in Berlin. Zweite durchgesehene Auflage. Pp. 175. Berlin: Verlag von S. Karger, Karlstrasse 15, 1920.

This is a manual not of psychotherapy, but of hypnotism for the physician, with just enough psychology to make it seem scientific and reasonable. When the historical part, general considerations and theories are left out, there remains a fair exposition of the methods, practice and results of a physician who constantly uses hypnotism. His technic is given and the reasons for it (and they are reasonable), the disorders in which he habitually uses hypnotism and comparison of this method with "persuasion." Incidentally, the author states that he has cured patients who had not been relieved by psychanalysis.

It is pleasant to note that the author insists that the operator must be a good physician first and a hypnotist afterward.

Any physician wishing to brush up on this therapeutic procedure will find the book satisfactory.

CLINICAL OPHTHALMOLOGY FOR THE GENERAL PRACTITIONER. A. Maitland Ramsay, M.D., Fellow of Royal Faculty of Physicians and Surgeons, Glasgow; Lecturer on Ophthalmology, University of Glasgow; Ophthalmic Surgeon, Glasgow Royal Infirmary; Author of "Atlas of External Diseases of the Eye," "Eye Injuries and Their Treatment," "Diathesis and Ocular Diseases," etc. With Foreword by Sir James Mackenzie, M. D., F.R.S. Pp. 476. London: Henry Frowde, Oxford University Press; Hodder & Stoughton, Warwick Square, E. C. 4, 1920.

In this book the author has abandoned the usual systematic presentation of particular diseases, adopting a less conventional, more discussive method, with the object of better meeting the needs "of the busy country doctor." For instance, instead of a chapter on glaucoma we find one on "The Clinical Significance of a Hard Eye." There may be advantages in speaking of the glaucomatous eye simply as a hard eye, but the lack of system is shown in considering first diagnosis and then indirectly etiology and pathology. For the general practitioner full consideration of the eye in relation to general disease would seem to be desirable but this has hardly been done in the brief treatment given choked disk, neuroretinitis, optic atrophy and arteriosclerosis. One is further disappointed in the lack of adequate attention given to the general factors in choroiditis and retinitis. The author is not wisely consistent in his advice to the general practitioner as to what to do and what not to do. He gives elaborate and minute details of procedure in the operation of iridectomy for glaucoma, but does not once say this operation should be done only by a skilled operator. On the other hand, he advises the practitioner not to do ectropion and entropion lid operations, which he might well perform.

On the whole, one must say that while the book is interestingly written and well illustrated with cuts and colored plates, and the busy physician could glean considerable useful information from it, a smaller book, with the subject matter arranged in a more scientific and definite manner, would better meet the needs of the general practitioner.

SURGICAL ASPECTS OF DYSENTERY, INCLUDING LIVER ABSCESS. By ZACHARY COPE, B.S., M.D., M.S. (LOND.), F.R.C.S. (ENG.), Surgeon to Outpatients St. Mary's Hospital; Surgeon to Balingbroke Hospital; Captain, R. A. M. C.; T. F. Surgical Specialist with Mesopotamian Expeditionary Force, 1916-1919; Hunterian Professor R.C.S. Cloth. Pp. 144. Oxford Medical Publications.

This work is based on the personal experience of the author and his associates in the Royal Army Medical Corps with the surgical complications arising from approximately 2,000 cases of dysentery, of both amebic and bacillary type. The subject is considered from three standpoints: (1) dysentery as a complication of surgical conditions, (2) various surgical conditions simulated by dysentery, and (3) surgical complications in the course of dysentery. He emphasizes especially the value of appendicostomy, cecostomy, or cecostomy with complete enterostomy in chronic unyielding cases, and warns against too great delay in operating. Surgeons operating on liver abscess are urged to keep a keen lookout for cerebral symptoms. He states that in dysentery abscess of the brain never occurs apart from liver abscess but that, according to Kartulis and Jacob, it occurs in 3 per cent. of liver abscess cases.